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EDITORS:
J. McCUNN, *F.R.C.S., L.R.C.P., M.R.C.V.S.*
R. LOVELL, *D.Sc., Ph.D., M.R.C.V.S., D.V.S.M.*
SIR THOMAS DALLING, *M.A., D.Sc., F.R.C.V.S., F.R.S.E.*

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EDITORIAL

BELIEVE IT OR NOT

THOSE who have perused the pages of the popular and medical press during recent months may have been seized with wonder and amazement. They have been able to read accounts of one of the greatest triumphs of modern science, i.e. the production of a vaccine which has been elaborated by Dr. J. E. Salk and his co-workers at the University of Michigan, and bids fair to help in the conquest of the dreaded disease Poliomyelitis.

Published almost simultaneously is a report on the medical use of hypnotism. Faith healing has also been in the news for it has been the subject of discussion between clergymen, medical men and certain enthusiastic laymen.

It would seem that the scientist, the mystic and the vendor of nostrums in the market place, have indeed something in common and that they are coming closer together. No doubt one may expect that in the near future some of the more erudite witch doctors of the African jungle will apply for admittance to the statutory register of medical practitioners and a rabbit's foot and a hypodermic syringe will not be incongruous companions in a modern doctor's handbag.

Professional men are the products of a very prolonged period of training during which they are encouraged to accept as fact only those things which allow of scientific proof. In consequence they are apt to be very stereotyped in their outlook and view with disfavour and some scepticism those things

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which do not conform to accepted orthodox scientific tenet. The history of medical art and science shows that the advancement of knowledge has not continued at a constant modulated metre but progress has been punctuated by a series of dramatic leaps and bounds, each of which has originated in some epoch-making discovery which has burst upon a wondering world. We know also that many of these notable and exciting events were the subject of much ridicule and condemnation by the orthodox workers of the period. Harvey encountered such difficulties when in 1628 he announced his discovery of the circulation of the blood and it is said that he delayed the publication of his treatise for a number of years because he feared that it might have an adverse effect upon his practice.

Great discoveries are rarely in harmony with current accepted opinion. If they were then they would not be unique and their advent would lose much of its revolutionary impact. Most men are averse to change; they prefer to follow an accepted path with its guarantee for their present and future safety. Fortunately there are always a few imbued with the spirit of adventure, who fret in any doctrinal yoke and find great zest in delving beyond the side of the road. It is from their ranks that most of the great inventors and discoverers have emerged. They are often classed as sophists and suffer the ridicule and contempt of their more placid and static friends, yet many have lived to see their ideas fructuate and excite envy and admiration. We know so little of the wonders of nature that nothing can be treated with contempt or ridicule until it has been subjected to critical test conducted without fear, favour or bias.

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GENERAL ARTICLES

KUMRI—SO-CALLED LUMBAR PARALYSIS—OF HORSES IN CEYLON (INDIA AND BURMA), AND ITS IDENTIFICATION WITH CEREBROSPINAL NEMATODIASIS

By J. R. M. INNES

Pathology Branch, Chemical Corps Medical Laboratories, Army Chemical Center, Maryland,

and C. PERUMAL PILLAI

Veterinary Research Laboratory, Peradeniya, Ceylon

Introduction

SUGGESTIONS were made [Innes, Shoho and Pillai (1952) and Innes (1951)] that *Kumri,* or so-called lumbar paralysis (an inaccurate label) might be identical with the disease now designated as cerebrospinal nematodiasis.

The name *Kumri* (occasionally spelled *Kumree, Kamree* or *Kamri*) is derived from Hindustani meaning a weak back. Such "stable" names in veterinary science are traditional, and may depict some graphic clinical sign, but they are puzzling and nebulous to medical workers, for few give any clue to the nature or cause of the disease concerned (c.f. scrapie, swayback, daft lambs, louping-ill, pulpy kidney and the staggers). Their worthlessness should demand their discard. but it is difficult, if not impossible, for this to happen.

We need not delve far back into the history of Kumri. All ancient references are quoted by Macalister (1917), to which frequent mention will be made; sixteen of the older papers cited by him were read by one of us (J.R.M.I.), and those by Gibbs, Skeavington and Molyneux date back to the early 19th century.* So, it would seem indisputable that this equine disease is of some antiquity, occurring no doubt from the time the British settled in India and imported horses for cavalry work, and then for racing and polo. From what Macalister says, and also Railliet (1895), the disease was not confined within India, but had been known by British veterinarians to occur in China, Malay States, Burma and Ceylon (and now we see it in Japan). If this is true, and if veterinary science had been developed adequately to facilitate proper disease investigation, then maybe it would have been recognised in other parts of Asia as well. That the disease was not, and still is not, rare is patent from the old literature, from what has been related to us by British and Indian veterinarians in India, and from Macalister's study which covered 23 autopsied cases, and the records on 18 horses in the Muktesar Laboratories (India).

*It was obviously well-known to Percivall (1843) and is mentioned in Vol. III of "Hippopathology," pp. 133-139, in a long discussion on worm-in-the-eye.

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Essays published during the 19th century on the disease are filled with contradictions and wild speculations on causation.* This was later perpetuated without any systematic attempt to tackle the primary principle of any disease study, namely, to identify its nature and thus define the problem. This disregard for a balanced pathologic approach to studies of disease is, however, an oft-repeated story in veterinary medicine—not only in bygone days in India.

However pathology is defined,† accurate observations will prompt some steps which can be followed subsequently, and a study of any lesion thus should be a object lesson, beyond mere description and diagnosis. Macalister's study is the only one in which the pathology of kumri was a considered object. Even so, it is of little service from what we know now because of confusing reasons which could not have been apparent to Macalister in 1917, or verily until many facts on cerebrospinal nematodiasis had been established. Yet, it is strange to contemplate that a possible solution was just round the corner, if eyes could see it. *Filariasis oculi* in horses—worm-in-the-eye or snake-in-the-eye—has just as ancient history as kumri, and many veterinarians in the past knew that one might follow the other, and both seem to have existed in the same areas. (See footnote about statement on Percivall in 1843.) More important still, filaria had been actually found in the nervous system, or cerebrospinal fluid, of kumri horses more than 100 years ago. Place (1911) and Saunders (1934) observed *Setaria* crawling in the spinal canal, and Saunders found worms within the cerebral meninges in one case, and in another a "cerebral granulomatous growth with eosinophilic infiltration suggestive of helminthic infection." Place disclosed a fresh facet in his work in Burma, and he was dogmatic on the ætiological role of filaria; he stated that not only horses were affected, but that in 200 cases in oxen, and 20 in male buffaloes, worms were found in the spinal meninges in every animal. Looking back, these were observations of cardinal moment marred only by lack of identification of a neurologic lesion to account for "paralysis," beyond mention of macroscopic softening, which pinpoints little about its true substance. In most papers, with pondering meditation on cause, there are tenuous arguments for and against a filarial ætiology, and that by Macalister embraces ten pages. Some of this is a little obtuse because of an inappreciation that there could be discrete helminthic infestation of a part of the nervous system without any

* "So whosoever shall entertain high and vaporous imaginations instead of a laborious and sober inquiry of truth, shall beget hopes and beliefs of strange and impossible shapes." —Francis Bacon, *The Advancement of Learning*, 2nd book, page 123, line 31. Edited by Aldis Wright, 5th Edit., 1926 impression.

† For those interested in this outlook, there is much for contemplation in the scholarly writings of Nicholson from 1920 onwards on "Studies in Tumour Formation" (Guy's Hospital Reports) and especially one on teratomata. We see then that pathology is a study of cause and effect with the intervention of time . . . but reaction is natural and physiological . . . since it is the only possible response of an organism . . . pathology is the science of physiological reactions of a living organism in an unnatural environment . . . changes from the old and usual, and therefore normal, to new and unusual and therefore abnormal state. The body, far from allowing itself to be moulded passively to new circumstances, alone determines the nature of reaction, and all the studies of microbe hunting in the world do not explain such vital phenomena.

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manifestation of visceral helminthiasis by the same brood of worms in the same animal. In cerebrospinal nematodiasis there is no such connection. Lingard (1896), who did much work on diseases in India, also believed in the filarial ætiology, and he may have introduced the use of antimonial and arsenical drugs (for surra) in the empirical treatment of kumri. (Place used santonin!)

Macalister gives the best clinical picture of kumri as painted by generations of British veterinarians in India. This accords with what has been seen in Ceylon, and related to us by others with experience in India. Macalister stated that the "great majority of kumri cases conform to a single type, presenting a crisply defined symptom complex, so that the term may be said to connote a single morbid entity. There are, however, variations; monoplegic and hemiplegic cases are known" (pp. 210-213). In standing, the hind legs are placed wide apart and tucked under the belly to achieve some balance, and the forelegs may be carried back in the same way. In walking, the animal has great difficulty in lifting its hind, or fore, feet, tending to drag the toes, and there is an exaggerated swinging out of the quarters. All those signs (not symptoms, mark you!) can be almost interpreted even from the still photographs of a kumri horse (see Macalister). Rather than early paralytic signs, balance is affected, and in initial stages there seems insufficient muscular power to lift the legs properly and move the body, but the animal is not "paralysed," least of all lumbar-wise. Signs of motor weakness and inco-ordination are emphasised when the horse is made to back or turn, and then there may be a whirling to the point of going down. Pressure on the loins may cause wincing and crouching, and the animal may suddenly collapse, and have difficulty in rising. Many older papers describe even milder varieties in which such signs did not progress, and yet other cases which became rapidly and severely ataxic and paralytic, and had to be destroyed. There is no fever, and so possibly the totality of the clinical picture, without any history of traumatic accident, helps to exclude the virus encephalitides if any of the latter were indigenous in the same locality as kumri. The reverse situation may have been more common, certainly in Japan, viz., that virus encephalitis could be confused with neural nematodiasis, and this may have been partly responsible for the latter remaining unidentified for so long. With the limitations imposed on exactitude of clinical neurological evaluation in horses, perhaps the described clinical condition could embrace a complex. We are not weaving a rope of sand, and glibly suggesting that all horses with weak backs and inco-ordination suffer from cerebrospinal nematodiasis. The same might follow as a result of physical trauma to the back, but the crucial yardstick of diagnosis could rest securely on a pathologic basis.

It is perspicuous that, clinically, kumri—with its variants of acuity in onset and tempo of course—is analagous to proven cases of cerebrospinal nematodiasis in sheep, goats and horses (whether in Japan, Korea or Ceylon). We cannot point to any major frank difference between kumri in horses and the disease of goats in Ceylon, including the seasonal occurrence. The prodromal stages may not be a true paralysis, but may be motor weakness

leading to inco-ordination and ataxia of either fore or hind limbs, or all four legs. In cerebrospinal nematodiasis this is fundamentally dependent on the locus and quality of damage to the nervous system by a worm, and only in the late stages may true paralysis be seen. We have mentioned elsewhere why there can be differences in clinical neurologic defects produced in horses, compared with smaller animals like sheep, by any destructive focus of the *same dimension.* We need only consider the great volume of brain and spinal cord in the horse (c.f. remarks on fibre tracts, Fig. 5). Finally, we can see no essential difference in the clinical signs in cerebrospinal nematodiasis in kumri, from those reported in the literature on "wobblers"* in horses in the United States [see Olafson (1942) and Jones, Doll and Brown (1954)]; pathological identity is another matter but full clarification has still to be achieved (see Jones, *et al.,* 1954).

There are two facts concerning the clinical history of kumri, which crop up repeatedly, although neither were emphasised by Macalister, yet they may be of pathognomonic import. The disease (kumri) has for long been annually enzootic in India, prevalent in some districts more than others, not seen in hilly parts, but in low-lying localities exposed to heavy, annual, seasonal rainfall. This was one of the first features noted by Crawford (for references to his work see Innes, 1951) many years ago, later also by Pillai, Mahamouth and Malkani, about the disease of goats in Ceylon. It is truly an annual enzootic (or epizootic) one, starting to occur in the fly season of the year, after a variable latent period. [It is important to mention here that in experimental work on cerebrospinal nematodiasis in Japan (by Itagaki, *et al.,* 1946-47) the range of latency period in horses was from 16 to 66 days between transmission of infective larvæ and onset of neurologic signs, and between 14 and 33 days in experimental goats (Ishii, *et al.,* 1953).] Under natural conditions some cases in horses, presumably, might not show signs until after the fly season, and which may have clouded the issue that the disease was strictly seasonal. In cerebrospinal nematodiasis, as the Japanese showed, this was related to the appearance of appropriate intermediate insect hosts as vectors for the infective worm larvæ. The second curious fact has been that in the area where the disease was first noted, it was found in newly-imported animals. Macalister intimated that kumri occurred firstly in Australian walers in India, only occasionally in Arabs, and that most of the native breeds were immune. Emoto (1928) dealt with the disease in goats imported into Japan from Switzerland where the disease did not occur. Later it was Corriedale sheep imported into Korea from New Zealand, and goats from India into Ceylon which were affected first. So, breeds which are truly indigenous, or which have been reared in those countries for generations, may have acquired some degree of resistance. These same phenomena are thus duplicated in proven cerebrospinal nematodiasis of sheep, goats or horses and in kumri; the long arm of coincidence can then become too stretched out for these

* Demonstrated to one of us (J.R.M.I.) by a cine film through the courtesy of Col. T. C. Jones, V.C., Armed Forces Institute of Pathology, Washington, D.C.; one clinical case was seen also in Maryland.

data to be of totally irrelevant significance, if borne along with ascertained facts on pathology.

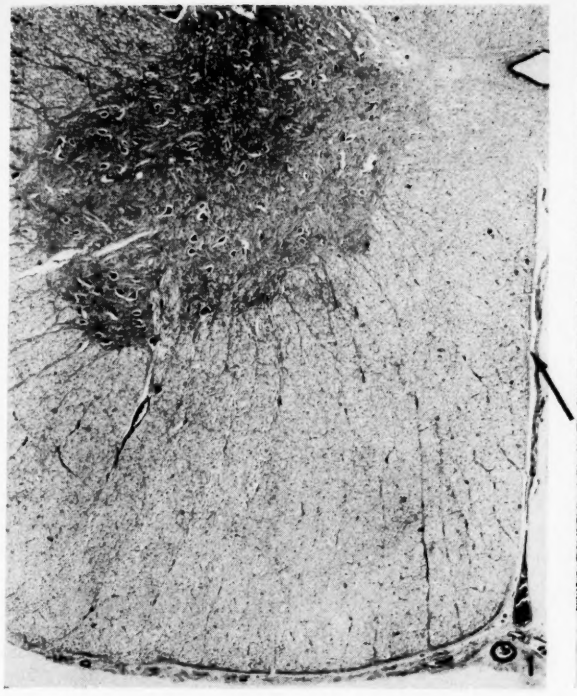
Definition of Cerebrospinal Nematodiasis

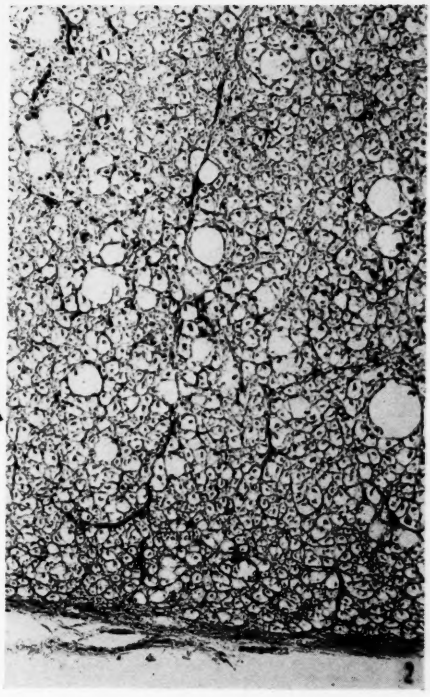
All studies, culminating in understanding cerebrospinal nematodiasis, starting with work by Japanese, were covered by Innes, Shoho and Pillai (1952), and it is necessary, in order to bring kumri into proper perspective, merely to summarise the salient features. The name was coined to designate an acute or subacute neurologic disorder of animals, which had occurred seasonally as an enzootic or epizootic in Japan, Korea and Ceylon, and where (like kumri) it was called lumbar paralysis. The concept, that the disease was caused by nematodes (*Setaria spp.*) stemmed from work by Japanese of the Korean Research Committee on Ovine Lumbar Paralysis (1939-1944). Clinically, the disease is no different from kumri, except that in sheep and goats there have seemingly been more cases with an abrupt onset of neuroparalysis, and not as in kumri with frequent cases showing prodromal motor weakness. The disease was defined pathologically as a focal malacia, with an inconstant concomitant patchy eosinophilic leptomeningitis. Foci might be located fortuitously in any part of the nervous system, but Shoho contended there was some predilection for the thalamus, cerebellum and spinal enlargement. It behoves us to stress again the difficulties sometimes encountered in determining naked eye changes, thus necessitating tedious histological study of the nervous system; in some Ceylonese goats the nervous system was sliced and embedded in paraffin, from the olfactory poles to the *conus medullaris*. The process is a true malacia, never blatantly bilaterally symmetrical, and sometimes an oblique tracking from the *pia mater* into and out of nervous tissue can be traced. Phagocytic glia predominate in the early stages: eosinophil leucocytic infiltrations may be prominent, and with the severance of nerve fibres in white matter there ensues acute Wallerian degeneration with its grossly swollen axis cylinders, which may be the most eye-taking feature of the lesion (so much so—Fig. 7—one wonders how it could have been missed). That all, or most, of this process is caused mechanically by a worm was proved by study of experimental needle wounds made in the spinal cord of sheep (J.R.M.I.). The disease was shown, firstly by Japanese workers, to be caused by *Setaria digitata* acquired by mosquitoes as microfilaria from the blood of the natural hosts (cattle) and later transmitted to abnormal hosts (sheep, goats and horses) as infective worm larvæ. After a variable latent period of migration and growing, some immature nematodes wander into the craniovertebral canal where they may penetrate brain or cord to produce lesions. This picture of pathogenesis, so admirably sketched out by the Japanese, begat new concepts into helminthic pathology, and this has engendered research which might profitably be applied to a future consideration of kumri. The Japanese referred to the disease first as "setariosis"; this was changed because we thought any nematode might produce the same pathologic result. This has been exemplified in work by Kennedy, Whitlock and Roberts (1952) and Whitlock (1952) on a disease of sheep, which they have called

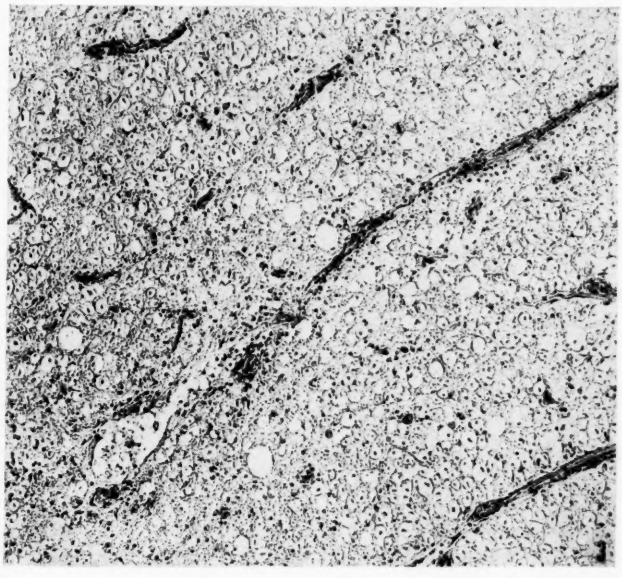
neurofilariosis, with an identical picture, but caused by a new species—*Neurofilaria cornelliensis*. The problem of clinically inapparent or masked helminthic invasion of the nervous system is important, and noteworthy observations by Tanaka, *et al.* (1945) included nine such cases in 100 horse brains examined. This should be correlated with studies by Beautyman and Woolf (1951) of a child with lesions of poliomyelitis in the brain stem, and with an ascaris larva in the thalamus, but no neurologic signs could be ascribed to the worm lesion, or if so they had been concealed by the virus infection. The conception that nematodes in the nervous system might facilitate, or exacerbate, neurotropic virus infections was later supported by experimental work of Mochizuki, Tominura and Oka (1954). They showed (1) if embryonated *T. canis* eggs were fed to abnormal hosts (mice), larvæ hatched and migrated from the stomach to the nervous system, but unless large numbers did so, they did not produce fatal paralysis caused by hæmorrhage in the brain [see also Tiner (1951 and 1953) and Sprent (1953)]; (2) if mice already fed *T. canis* eggs were inoculated subcutaneously three to four days later with a Jap B encephalitis virus (an attenuated strain which did not produce encephalitis after subcutaneous injection) then a significant number died from acute virus encephalitis in a few days. The mechanism of this adjuvancy between worm and virus is unknown.

In any probing into a helminthic cause of a malacic disease, it is advisable to accentuate intrinsic difficulties found by the Japanese, and encountered in the caprine disease in Ceylon (and now in the equine kumri). Immature worms may not be found in, or near, the malacic lesion by the time an affected animal is autopsied, especially if the duration of the disease has been weeks. There might even then be only one worm. It may wander away, be found on filtering the cerebrospinal fluid, or it may travel out of nervous tissue after fixation and be found in the latter fluid. In chronic cases, the worms may have died, become calcified, or only barely recognisable fragments of a hyaline capsule may be found in sections. To find a worm might be luck, or like seeking the proverbial needle in the haystack, and more so when dealing with a brain and cord as large as that of the horse. The parallel difficulties of Wilder (1950) in her work on nematode endopthalmitis in children can be reiterated; in one case 2,300 serial sections were made of a whole eye before remnants of a worm were found. Confronted with all facts, it is easy to be sagacious in retrospect, and understand why this disease, known as kumri in India before 1800, escaped true recognition of its nature and cause. Locating a lesion and identifying a worm might demand meticulous, humdrum, time-consuming study, while its omission might lead to erroneous impressions, and leave perplexity in comprehension why animals could have such varied neurologic signs without tangible evidence of effect on nervous tissue and of cause.

There are good inferential data denoting a variety of events which could follow the entry of a worm into the craniovertebral cavity. (1) The worm could wander in the column of spinal fluid, never penetrate nervous substance and produce simply meningitis and radiculitis, which could explain cases with recoverable clinical signs of motor weakness. (2) The worm could invade the







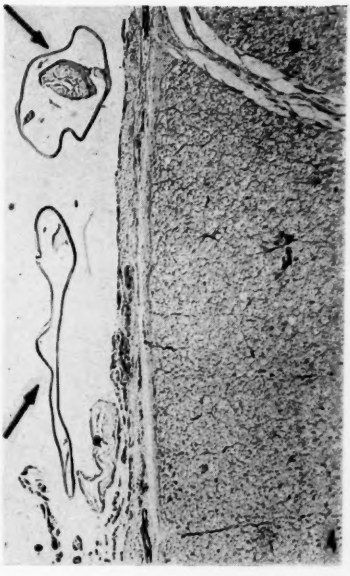


Fig. 1. *Kumri spinal cord, horse. Case* 1. About C.6-7; central canal top right and ventral septum right; this represents also the state of the same level of spinal cord from Case 2; showing absence of any changes, except minor degree of secondary degeneration (arrow), not too distant from lesion in C.8 (Fig. 3). Hæmotoxylin eosin. x 15.

Fig. 2. *Spinal cord. Case* 1. A level still more caudal than that in Fig. 1 (about C.7) around 2 cm. above actual lesion; ventro-lateral white column with *pia mater* at bottom; this appearance may be seen above or below a malacic focus, representing Wallerian degeneration seen as scattered empty blown-up "holes" in paraffin sections; these holes may represent fibres in which the myelin has been liquefied, and swollen broken-up axis cylinder dropped out, or the latter cut through areas between fragments. In this cord scattered degenerate fibres were present in ventro-lateral columns more on one side than the other, and a few in the dorso-lateral columns. Hæmotoxylin-eosin, x 200.

Fig. 3. *Spinal cord. Case* 1. Ventro-lateral column at level between C.8-T.1. Irregular area of white matter with thickened vessels and septa, loss of myelin fibres through malacia, increase of reticulum and glial fibres. Patchy areas of this lesion were scattered round into dorso-lateral columns almost to tip of dorsal grey matter. Hæmotoxylin-eosin. x 100.

Fig. 4. *Spinal cord. Case* 1. Same level as Fig. 3, showing parts of a worm (arrows) cut obliquely, and lying on, but outside, *pia mater* (left). Hæmotoxylin-eosin. x 100.

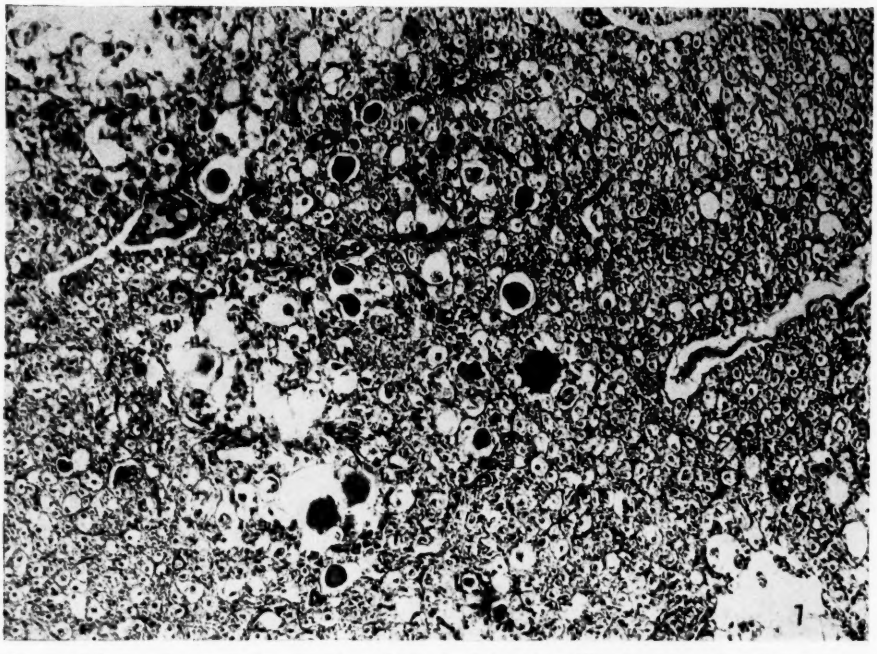


Fig. 5. *Spinal cord, horse. Case 2.* About C.8-T.1 level through middle of lesion in ventro-lateral columns of white matter, central canal top right, ventral septum at right. Irregular oblique focus tracking in from *pia* with microcavitation. Doubt has been expressed by some regarding the clinical significance of such a lesion. There are around 20,000 fibres in the area figured in ventro-latera' columns of normal equine spinal cord—C.8—and seen transversely. In the area near right arrow in this picture around 80 per cent of fibres have been destroyed. Hæmotoxylin-eosin. x 15.

Fig. 6. Higher magnification of subpial area (left arrow from Fig. 5) showing the marginal area of malacia extending round ventro-lateral column; some phagocytic glia present. Hæmotoxylin-eosin. x 200.

Fig. 7. Higher magnification of area mapped in square of Fig. 5. Showing destruction, to point of disappearance, of nerve fibres by liquefaction and microcavitation; note fantastically swollen axis cylinders as sequel to acute Wallerian degeneration; phagocytic glia are present but not numerous. Hæmotoxylin-eosin. x 200.

(*Article by Innes and Pillai, page 223*)

nervous system, produce a lesion in a non-vital part, or one which could be clinically silent (e.g., cases in horses by Tanaka, *et al.*). (3) The worm could produce the classic malacia responsible for acute ataxia and paralysis. (4) One worm could produce more than one lesion by irregular repeated burrowings. (5) More than one worm could produce multiple lesions, which need not all be of the same age. (6) An animal could be reinfected in the same or different seasons (Shoho, and Tanaka, 1955), none of which need be fatal, for clinical outcome depends on the situation and amount of damage in the brain or cord. All the above emphasise the complexities of pathology and pathogenesis, and help explain the clinical variants.

Shoho (1952 and 1954) conducted two separate field trials in Japan, and produced evidence that a filaricide—1-diethylcarbamyl-4-methyl piperazine (trade name: Caricide, Lederle, New York)—had some prophylactic value, while in mild cases if the drug was given before the theoretic fulmination of a true destructive malacic lesion, it might also have some curative effect.

Study of Kumri

Four equine cases were obtained in Ceylon, three belonging to the same person who had lost at least 15 riding ponies from the same disease in 15 years. Two horses were autopsied; two, obtained at an early stage, responded dramatically to therapeutic treatment with caricide and will be reported later. In Ceylon no other encephalitis besides rabies is known, so clinical confusion is rendered more remote.

Clinical History, Case 1. (P.M. 45/54.) Aged Arab. Motor weakness of hind quarters noted two months before first clinical examination by C.P.P.; animal probably affected about August-September; signs did not progress, but no improvement. Animal also suffered from *Filariasis oculi,* surgery for removal of worm leaving a linear scar. Weakness of hind limbs; fore limbs carried back, hind feet incompletely lifted with dragging of toes. At a trot the legs and quarters swung widely out; a straddled type of turning and with a tendency to fall. No loss of condition or appetite; no wasting of muscles; no loss of sensation; micturition and defœcation normal. This case showed no clinical difference from kumri seen in India. Animal killed by intravenous chloral hydrate in January, 1954, and thus about four months' duration.

Clinical History, Case 2. (P.M. 186/54.) Aged Arab stallion, same owner as Case 1. Observed to be weak in the back January 5, 1954. By the 12th all signs described for first case had advanced acutely and ataxia marked. On the following day it could not be moved without risk of falling; animal down and died on the 16th.

Pathology

Remarks are confined to the nervous system, all other findings in the viscera being without relevant significance.

Methods

In both cases the brain and entire length of spinal cord were taken out intact with meninges and fixed in formalin saline. The brain of each case

was sliced coronally, and representative blocks were taken for sections. Neither macroscopically, nor microscopically, were any suspicious lesions noted in the brain, and all further data refer to the spinal cord, the latter measuring approximately 60 inches in length. The attached dura was slit, removed and preserved. No worms were found attached to dura or cord. The cord was sliced at each segment from C.1 to S.1-2, and at least one transverse cut was taken from each segment for histological preparation. All areas with suspicious gross changes were noted; after checking with the histology, the interpretation that any gross changes were present was wrong on each occasion! After a survey of sections made from all levels (some 36 blocks) in areas where any changes were found, the whole of the remaining segment of cord was cut for further sections, and from some of these blocks serial sections were made. In each horse thus at least 60 blocks from the spinal cord, and a far larger number of sections, were cut and stained by hæmotoxylin-eosin, and a myelin, a glial or a reticulum method.

Distribution and Nature of Lesions in the Spinal Cord

In Case 1 a lesion was found in the initial block cut from C.8, and was traced through the remainder of 13 blocks cut from the cervical enlargement, gradually fading into normal tissue by about T.2. In the second case the location of the lesion was approximately the same, but it extended through fewer blocks from C.7 to T.1.

The nature of the entire changes found had in general an identical appearance to those described in cerebrospinal nematodiasis [Innes (1951) and Innes, Shoho and Pillai (1952)]. Much of the pathology can be understood without circumlocution now, by reference to these papers and to the present photomicrographs. It is left to stress a few points. In each case, in the survey of the entire cord, a number of scattered tissue alterations were found which could be important or incidental. Focal lymphocytic infiltrations were found dotted in many parts of *dura mater,* not so frequently in the leptomeninx, often in nerve roots, but rarely in spinal ganglia or *ligamentum denticulatum.* Isolated *corpora amylacea* in *dura mater* were noted. Perivascular hæmorrhages were sometimes seen, but never gross hæmorrhage in or near any lesion in the cord. Eosinophil participation in cellular infiltrations was never prominent, and often absent.

In both cases the focal lesion in the cord was confined to the ventro-lateral white matter, and in each horse was more severe on one side than the other. A composite picture would represent the damage as being an irregular, obliquely, longitudinal and streaky patch starting under the *pia* in the ventral column, piercing the white matter for some distance on one side, and in parts overlapping the adjoining ventral white matter of the other side. Grey matter in all parts of a cord was normal, the motor ganglion cells always being well stained. There were no inclusion bodies present. The lesion was not comparable with any seen in the infective encephalomyelitides.

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The quality of the lesions in the two cases is considered separately, for there was a difference in amount and stage of damage and in tissue reaction. This can be related to one case being of four months' duration, and seemingly clinically static when killed, while the other had been acutely progressive for two weeks. The second case can be dealt with first in precise terms and helped by examination of the figures 5-7. All features of the classic nematodal malacia, as described previously, were present in the irregular discrete focus in the cord. The malacia, the liquefaction with streaking microcavitation, and the fantastic gross swelling of axis cylinders representative of Wallerian degeneration were unmistakable. As serial sections were examined consecutively from a cranial to caudal direction, the lesion faded into normal white matter, but secondary degeneration could be made out beyond that, represented by blown-up holes (see Fig. 2)—the end-result of fibres which have been wholly liquidated (compare with Figs. 4 a-d of needle trauma of the cord, Innes, *et al.*, 1952).

In Case 1 the focus in the cervical enlargement showed some streaking liquefaction, crevices of microcavitation, and evidence of secondary degeneration at the margins and also in cranial and caudal directions. The process had passed the acute stage; there was loss of myelin fibres, reactive glial scarring, and markedly thickened vessels and septa with increase of reticulin around. Cellular infiltration, mainly lymphocytes, was not severe, and there were no changes in the grey matter. The changes were comparable to the older chronic lesions described by Shoho and Tanaka (1955).

No worms were found in the *depths of the lesion* in either case, after a search of very many sections. In Case 1 parts of an unidentified helminth were found, cut obliquely and lying on the *pia mater* at C.8 at the margin of the lesion described; and two immature worms were found in the cerebrospinal fluid from the same case (by C.P.P.) but were not identified. No worms were found in the 150 cubic centimetres of spinal fluid collected from the second case. Emulsions of brain substance from this animal were injected intracerebrally into guinea pigs, rabbits and mice with negative results.

Discussion

The clinical picture, history and annual seasonal occurrence of kumri in Ceylon (by inference we include the disease in India) dovetail with all those aspects of cerebrospinal nematodiasis. The pathology of kumri has been demonstrated, at least tentatively. The lesion in the spinal cord of the two cases falls into the process pattern of a focal malacia which can be initiated by nematodes. In one case, a worm was found near a lesion and other unidentified worms were found in the cerebrospinal fluid. Although evidence is still unavailable which points to an unequivocal binding association between cause (worm) and effect (focal malacia), let us not be blinded by dictates of science. Our main contention is that there are inescapable data which betoken the way for an intensive study of kumri, along paths with the thorny way somewhat cleared and signposted. The disease is worthy of study from several

angles; the fact that it is not a rarety, but enzootic, in horses which might have much financial value (Arabs, thoroughbreds, or polo ponies) is one. [If serious reflection is given, kumri might be far more important than many other equine disorders, upon which much money has been spent on research with far less productive result.] Cerebrospinal nematodiasis is an extremely fascinating biological problem, not only a veterinary one, for it is a complex puzzle involving basic host-parasite relationships. Penultimately, the article is published to stimulate those who control the financing of research, and others who can instigate and pursue studies which now necessitate the closest collaboration between a pathologist, a helminthologist and an entomologist—all working in India and Ceylon. Identification of the items which await inquiry, then, needs no imaginative ingenuity. More cases in horses require to be studied from clinical and pathologic viewpoints. The filaria, which have been found so often in kumri cases (and in equine eyes) in India (and Ceylon) require accurate species identification and more clarification of the synonyms used for the species of *Setaria* is required. Textbooks on veterinary helminthology are most confusing and of no help in any of these directions. The natural mammalian hosts of *Setaria spp.,* in all lands, could be better established, and the intermediate insect hosts for *Setaria* in India and Ceylon must be found. The work of the Japanese on the species of mosquito involved, and the statement by Chandler (1950), that *Stomoxys* may be implicated in the life-cycle (in the United States), should be remembered.

Viewed in the above light, the lack of finding a worm in the second case is not an irrevocably serious omen—success would have bolstered convictions. In any considered thought on kumri, with its postulated complex pathogenesis, and on the evidence produced, we realise it is impossible to proffer absolute proof that there is a connection between (*a*) worm, (*b*) neurological signs and (*c*) lesions in the nervous system. But we cannot employ the postulates of Koch (which in any case are not laws like those of the Medes and Persians), appropriate for infectious diseases, to disorders of this nature, which are incapable of experimental transmission to laboratory animals. In the first place, accurate observation of the disease under natural conditions is essential. Mere presence of a worm within a lesion in the nervous system does not mean it was the cause, and the fact that a worm may wander away from a malacic focus cannot be proved by what may be seen finally in a dead horse. The casual worm, unfortunately, if found in the spinal fluid or in a lesion, cannot be taken and used for experiment in another animal. One might proceed further and say that after maturation from microfilaria, if infective larvæ are introduced by a hypodermic needle into an animal, we cannot be certain these are the same parasites which will be found later causing damage to the nervous system. The problem with its labyrinthine complexities is therefore realised and posed by us, and there is no easy way to follow except along the paths of experimentation successfully trodden by Japanese workers [e.g., Itagaki, *et al.,* and Ishii, *et al.* (1952) and others before them]. Collection of clinico-pathologic data on a large series of kumri horses showing constant association between worms and

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focal malacias will give a truer picture, maybe disentangled from the presence of other clinically confusing diseases.

It is necessary to refer to the findings of Macalister on the pathology of kumri, and we must be vindicated from any accusation of carping criticism. In substance, his descriptions and illustrations of the pathology are virtually of no benefit, and there is no justification for his naming the disease as "combined diffuse sclerosis and central poliomyelitis"—which had best be forgotten. Macalister's summary of the fibre tracts in the white matter of the cord do not apply to horses, for nothing is known about the equine long fibre tracts, beyond that the pyramidal tracts as motor paths are probably of little significance. None the less there can be no argument that the ventro-lateral columns, affected in our cases, do contain the motor tracts. Like the sheep, pyramidal tracts in horses probably fade out about the upper cervical levels, and the rubro-spinal paths may be more important—facts pointed out by Sir Fred Smith in his "Veterinary Physiology" fifty years ago. In fairness to Macalister, his pathologic studies were apparently not completed.

Little discussion of the pathology is required, this having been done in connection with cerebrospinal nematodiasis. A little caution may be inserted at this place. All focal malacias which might occur in the animal nervous system—for example, there are the rareties caused by vascular catastrophe or occluding emboli—manifestly cannot be deduced to be caused by worms, and we possess examples of such lesions. Yet, discrete isolated asymmetrical foci of softening might be highly suggestive. Bilaterally symmetrical malacia, with some tendency towards neuroanatomical selectivity, does not fit into the picture of cerebrospinal nematodiasis, and such diseases have now been reported in animals. Cordy (1954) described a disease of horses, with symmetrical areas of softening in the *globus pallidus* and *substantia nigra,* and produced experimental evidence that it was caused by a variety of California thistle. Plowright (1954) and Innes and Plowright (1955) defined a peculiar focal symmetrical spinal poliomalacia causing paralysis of sheep in the Rift Valley of Kenya. Hartley (1954) in New Zealand has identified symmetrical malacic lesions in sheep occurring in the basal ganglia and midbrain, and a cortical variety in pigs. Neurologic diseases in animals seem to blossom from all lands.

In conclusion, we might return to the problem of filarial cause in kumri, first suggested over 130 years ago, known perhaps before then, and reiterated so often, but not based on good pathology, by subsequent observers. Some reasons have been given for the possible confusion, and why some workers could not (or would not) believe the visual evidence. The problem of *filariasis oculi* has often been brought into line with kumri, but what has never been visualised is that probably both need be no more than part of a helminthic neuro-ocular complex, and that it could be a haphazard accident, in nematodal migrations in the animal body, which decides whether their final resting abode is the eye, brain or spinal cord. Innes and Shoho (1953) dealt with the idea that the nervous system may be a peculiar locus of attraction for worm wanderings in

abnormal hosts. There has been no investigation into paths of tissue migration taken by filarid worms to reach the eye in horses [and the same applies to the human endophthalmitis described by Wilder (1950) in children], but intravascular paths for larval migration need not be the only explanation. Although there may be some random chance in worms being located in any particular part of the nervous system, yet there may be some selectivity once they arrive; in both these equine cases, within the limits of examination imposed by time and labour in section cutting, the focus was present in the cervical enlargement of the cord (see also findings of Jones, *et al.*, 1954, in the case of wobblers).

Summary

Four cases of kumri in horses were studied, two of which responded in a dramatic fashion to treatment by a known filaricide and two horses were autopsied, the brain and spinal cord being examined with care. The clinical history with the signs of motor weakness, inco-ordination, ataxia and paralysis, and then the annual seasonal incidence are analagous with cerebrospinal nematodiasis in sheep, goats and horses (Japan and Korea) and goats (Ceylon). The pathology of kumri was identical with the focal malacia following invasion of the nervous system by nematodes. The filarial cause of kumri, which is a theory of ancient vintage, was discussed, and is of ample substance to merit further detailed studies of the disease by pathologists, helminthologists and entomologists. More evidence is accruing to indicate that cerebrospinal nematodiasis may be a biological problem of global importance.

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REFERENCES

Beautyman, W., and Woolf, A. L. (1951): *J. Path. Bact., 63,* 635.
Chandler, A. C. (1950): "Introduction to Parasitology (with special reference to parasites of man)," Ed. 8. New York: Wiley & Sons.
Cordy, D. R. (1954): *J. Neuropath. exp. Neurol., 13,* 330.
Emoto, O. (1928): *Schweiz. Arch. Tierhlk., 69,* 297.
Hartley, W. J. (1954): New Zealand. Personal Communication.
Innes, J. R. M. (1951): *Brit. vet. J., 107,* 187.
Innes, J. R. M., Shoho, C., and Pillai, C. P. (1952): *Brit. vet. J., 108,* 71.
Innes, J. R. M. (1952): *Indian vet. J., 29,* 81.
Innes, J. R. M., and Shoho, C. (1953): *Arch. Neurol. Psychiat., 70,* 325.
Innes, J. R. M., and Plowright, W. (1955): *J. Neuropath. exp. Neurol. 14,* 185.
Ishii, S., *et al.* (1953): *Brit. vet. J., 109,* 160.
Itagaki, S., Taniguchi, M., and Kawata, M. (1946-47): *J. Jap. vet. Med., 3,* 79, *4,* 73 and 105.
Jones, T. C., Doll, E. R., and Brown, Ross (1954): *Proc. Ann. Cong. Amer. Vet. Med. Ass.* Seattle.
Kennedy, P. C., Whitlock, J. H. and Roberts, S. J. (1952): *Cornell Vet., 42,* 118.
Macalister, G. H. K. (1917): Memoirs Dept. Agric. in India, Vol. 11, No. 8, pp. 207-261.
Mochizuki, H., Tominura, T., and Oka, T. (1954): *J. Infect. Dis. 95,* 260.

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Place, J. (1911): *J. trop. vet. Sci.*, *6*, 44.
Plowright, W. (1954): *Vet. Rec.*, *66*.
Olafson, P. (1942): *Cornell Vet.*, *32*, 141.
Railliet, A. L. J. (1895): "Traité de zoologie médicale et agricole," Ed. 2, p. 507. Paris: Asselin and Houzeau.
Saunders, P. T. (1934): *Ann. Admin. Rept., Madras* (India), *Civ. Vet. Dept.* (Paraplegia in horses).
Shoho, C. (1952): *Brit. vet. J.*, *108*, 134.
Shoho, C. (1954): *Vet. Med.*, *49*, 459.
Shoho, C., and Tanaka, T. (1955): *Brit. vet. J.*, *111*, 102.
Sprent, J. F. A. (1953): *J. Infect. Dis.*, *92*, 105 and 114.
Tanaka, T., Suu. S., Shoho, C., and Yamagiwa, S. (1945): *Jap. J. vet. Sci.*, *7*, 117.
Tiner, J. D. (1951) *J. Parasitol.* (No. 5, Pt. 2), *37*, 21.
Ibid. (1953): *J. Mammal.*, *34*, 153.
Whitlock, J. H. (1952): *Cornell Vet.*, *42*, 115.
Wilder, H. C. (1950): *Trans. Amer. Acad. Opthalm.*, *55*, 99.

THE ECONOMIC SIGNIFICANCE OF THE INCIDENCE OF MORTALITY IN FOWL

By R. COLES, B.A., Ph.D., M.Sc.(Econ.), M.Sc.(Agric.)

Ministry of Agriculture, Fisheries and Food

THE value of the fowl is small compared with that of the larger farm animals and therefore attempts at prevention of disease are more common than the curative treatment of sick or ailing birds. Since chicken are carried on the great majority of farms, and very often in extremely small units, it is not surprising that conditions under which they are managed vary, and that on many farms the conditions are very poor. In consequence, mortality rates in some flocks are high. Various estimates have been advanced on the financial losses sustained by the poultry industry from mortality from all causes. Before the war an estimate of this cost, put forward by the late A. J. Macdonald, of Harper Adams Agricultural College, was four million pounds with an industry the output of which was then valued at 48 millions. With a total output valued to-day at 180 million pounds, the same rate of mortality would result in an annual loss of 15 millions.

While a high average level of mortality is believed to be the cause of heavy monetary losses to the poultry industry, it is extremely difficult to give a reliable estimate of the actual loss sustained, or the major causes of such losses. On individual farms the picture is distorted by frequent sales of stock, by additions at various ages, and by culling at varying degrees of intensity. Culling is carried out on most farms to remove a bird which seems likely to prove a poor layer or one which appears likely to succumb to disease. In both instances judgment is based on visual observation and may or may not be correct. This common practice on the part of poultry farmers may, in fact, be the cause of some error in arriving at an assessment of the relative importance of

certain diseases. The analyses of post-mortem reports from the laboratories of the Ministry of Agriculture and other organisations have been employed to provide some indication of this relationship, although it is realised always that the number of carcases examined at these centres are not necessarily a fair sample of the number of birds dying in the country. According to these figures (Coles, 1954) the so-called avian leucosis complex is responsible for about 25 per cent of deaths occurring with adult stock and coccidiosis continues to be the major cause of death with younger birds. Yet it is known that many breeders of repute who send carcases for post-mortem examination, where the cause of death eludes them, are content to cull and kill birds described by them as "going light," weak or poor "doers" without further investigation. It is probable that many of the birds so described are affected by leucosis, and such actions contribute to the difficulty of assessing accurately the relative importance of some diseases.

In default of much data being secured from poultry farms, some reliance has been placed on the records of laying trials. In these trials, picked samples of birds from a number of farms are submitted and detailed records of the unculled birds over a laying year, generally of 48 weeks, are available. Unfortunately these trials with one exception do not include records of mortality of young stock, and, since selection of the trial birds is practised, it cannot be contended that the birds are a fair sample of the individual breeder's birds. Dealing with the mortality at such trials Dudley, Dobson and Gordon (1941) drew attention to the rise in mortality from 6.24 per cent in 1929-30 to 17.24 per cent in 1936-37. Bearing in mind Macdonald's opinion of the cost to the industry of mortality in the immediate pre-war years, it will be appreciated that, if these variations in mortality at trials are reflected throughout the industry as a whole, the financial losses from mortality can vary in importance over relatively short periods, and that mortality among flocks can become a main factor influencing the economics of the industry.

The figures for the major cause of mortality shown in the paper by Dudley, *et al.*, for both county and open laying trials are roughly in line with those from an analysis of current post-mortem examinations at official laboratories. In the paper by Dudley, *et al.*, it is shown that "lymphomatosis and allied disorders" are responsible for most deaths, viz., 32.9 per cent at county and 23.7 per cent at open trials; disease of the reproductive system is the second most important single disease entity.

Mortality figures obtained from laying trials in recent years show no abatement of the high death rate reported by Dudley and his co-workers in 1936. During the last four years the percentage mortality rate at two of the largest trials has ranged from 9.1 to 14.58 over a 48 weeks trial year. At one of these trials, where chicks are hatched and reared, the mortality rate from day old to point of lay has varied from 2.06 to 6.4 per cent. Several poultry keepers have in recent years drawn attention to the heavy financial losses which mortality rates must inflict on the poultry industry, but no precise information has been submitted. This failure is no doubt due to

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the common practice already referred to of culling, selling birds and adding to the flock throughout the year, so that an accurate record of the percentage dying is impossible to obtain. In particular, no reference has been made in any account of the death rates at laying trials or elsewhere to the time in the life cycle of the bird when mortality occurs. This is a point of great economic importance, for the bird which dies at five days old has cost the poultry farmer little more than the cost of the chick, while the bird which dies at five months has cost the owner the rearing costs, which will include about one-third of a hundredweight of food, and, at this stage, the bird has probably contributed nothing in the way of eggs to offset these heavy charges. By contrast the bird which dies at 18 months, and which has laid reasonably well over its pullet year, probably leaves a margin of profit. Since such a bird may probably have been culled at the end of the season in any case, the only loss may be that resulting from a reduced or non-existent carcase value.

While popular opinion accepts the view that mortality in the national flock is a heavy debit item, there is also a popular belief that mortality levels are higher with late hatched chicks. Kennard (1933) has produced, also, evidence showing that pullet-bred pullets manifest a higher level of mortality than hen-bred pullets. This view is widely accepted, as is also Kennard's view that the mortality rate from fowl paralysis is lower in the hen-bred pullets than in pullet-bred stock. It is also a common belief that a high death rate—particularly during the rearing period—is correlated with a high death rate from lymphomatosis, although evidence submitted by Waters (1951) does not support this popular belief so far as mortality rates with adult stock are concerned.

Experimental

(1) *Incidence of Mortality.*

With a view to attempting to secure precise evidence on the incidence of mortality in the life cycle, and particularly the economic implications, detailed records were kept of a flock of White Leghorns over a period of four seasons each of 78 weeks. The majority of the birds were pullet-bred, but each year a number of hen-bred pullets were included and carried in the flock without segregation. Hatching took place each year over an approximate 60-day period, with the first hatch coming off about March 1, and the last around the end of the first week in May. Apart from the successive hatches being brooded in separate groups over the first six weeks, the birds were not segregated during the rearing or laying periods. Each hatch was carried over a period of 18 months from date of hatching. No culling was practised and all birds dying or killed *in extremis*, were subjected to post-mortem examination. All birds were trapnested from first egg until December 31, of the year in which they were hatched, if indeed they had laid by the latter date. After December individual recording was not generally practised. All birds were reared out of doors after the first six weeks and carried on range in colony houses. Only those groups of cockerels selected on the basis of their sisters' performance up to December 31, were

retained after that date, and so no data of the mortality among cockerels are presented.

Of the total number of 4,181 pullet chicks hatched, 922 or 22.05 per cent died before they were 18 months old. Figures for the successive seasons

TABLE 1

Incidence of mortality in a flock of White Leghorns over 4 seasons each of 78 weeks duration.

Season	No. of pullet chicks started	Total No. dying		No. dying from Fowl Paralysis & Leucosis	
		No.	%	No.	%
1950/51	811	247	30.46	141	17.38
1951/52	1,235	311	25.18	152	12.31
1952/53	992	199	20.06	92	9.27
1953/54	1,143	165	14.43	41	3.59
Total all seasons	4,181	922	22.05	426	10.18

are shown in Table 1. An analysis of the causes of death revealed by post-mortem examination is set out in Table 2. From this Table, it will be noted that about half the deaths are attributable to one or other of the several

TABLE 2

Analysis of Post-mortem Examination results over four seasons each of 78 weeks

	1950-1	1951-2	1952-3	1953-4	4 seasons
	%	%	%	%	%
Disease of Reproductive System	3.6	4.8	9.04	9.09	6.2
-do- Digestive	8.1	3.2	10.6	6.7	6.7
-do- Respiratory	4.5	4.8	5.02	7.3	5.2
-do- Urinary	2.0	5.1	2.01	6.06	3.8
-do- Circulatory	1.2	1.3	2.01	2.4	1.6
Disease of Liver	4.0	4.5	3.02	3.6	3.9
General emaciation	-	1.3	1.01	-	0.7
Prolapsus of Oviduct or Intestines	-	0.3	-	-	0.1
Undiagnosed infection	-	-	-	2.4	0.4
Undiagnosed or decomposed specimens	9.4	8.04	7.5	11.5	8.9
Injury	0.4	0.9	2.5	1.2	1.2
Leucosis and Paralysis	57.1	48.9	46.2	24.8	46.2
Cysts and tumours	1.2	3.2	3.5	0.61	2.3
Coccidiosis	8.5	12.9	8.5	23.03	11.5
Vent Gleet	-	0.3	-	1.2	0.3
Salmonella infections	-	0.3	-	1.2	0.3

manifestations of the so-called leucosis complex. This level of mortality from leucosis of 10.18 per cent, or about half the total, is in excess of the figure reported by Coles (1954) of about a quarter of all causes of adult mortality

found at official laboratories. It should, however, be borne in mind that a substantial number of the deaths from leucosis found in this investigation occurred before maturity. Attention is also directed to the author's earlier remarks that many poultry keepers cull heavily birds which are "going light" and do not subject them to post-mortem examination because they wish to sell a carcase which is still saleable, and because they assume that leucosis is the cause of the symptoms. It has already been suggested that, on these grounds, the actual percentage of deaths attributable to leucosis may well be in excess of the approximate 25 per cent cited, and the latter should not be regarded as the true figure.

It should be stated that when fowl paralysis first manifested itself in this closed flock in 1948, no attempt was made to control it by culling or rearing in isolation, but an endeavour made to breed for resistance. As a consequence

TABLE 1

Incidence of mortality attributable to Fowl Paralysis and Avian Leucosis and percentage assigned to each.

Season	Death rate caused by Paralysis & Leucosis	Percentage caused by Fowl Paralysis	Percentage caused by Leucosis
	%	%	%
1950/51	17.4	86.9	13.1
1951/52	12.3	90.3	9.7
1952/53	9.2	88.5	11.5
1953/54	3.6	91.7	8.3

the incidence of the disease may be above normal in 1950-51, and below average in 1953-54, but it is believed that the average over the five-year period approximates to the average incidence found in the national flock.

Recently a trend of thought has developed which believes that some diseases are wrongly classified under the term "avian leucosis" (Campbell, 1954). This view, while not accepted by all workers, appears to be gaining a good deal of support and would regard fowl paralysis as outwith the so-called avian leucosis complex, and visceral lymphomatosis as one of the diseases to be included under the term avian leucosis. In this flock, those birds dying from what has, until recently, been described as neural lymphomatosis (fowl paralysis) formed the majority of birds dying from one or other manifestations of the so-called leucosis complex. In view of the development of the opinion described, the number of birds dying from fowl paralysis and avian leucosis including visceral lymphomatosis) is shown as one figure in Table 2 but separately in Table 3. Although the number of birds in this experiment succumbing to what has been described as the avian leucosis complex has shown a steady decrease from 17.4 per cent to 3.6 per cent, the proportions dying from fowl paralysis and visceral lymphomatosis have remained remarkably constant. The programme of breeding for resistance described elsewhere

Coles (1954) appears to have been equally successful against both diseases, which are now increasingly regarded as being quite separate disease entities.

For every bird dying, a note has been taken of the number of weeks elapsing between the bird's hatching date and date of death. It has thus been possible to show for any one season, and also for the total number of seasons, the number of birds dying at any particular period throughout 78 weeks. The cumulative incidence of mortality at successive weekly stages over 18 months for the four seasons is shown in Fig. 1, and for each of the four seasons in Fig. 2. In all figures the cumulative deaths due to fowl paralysis and avian leucosis are shown in addition to the figures for deaths from all causes.

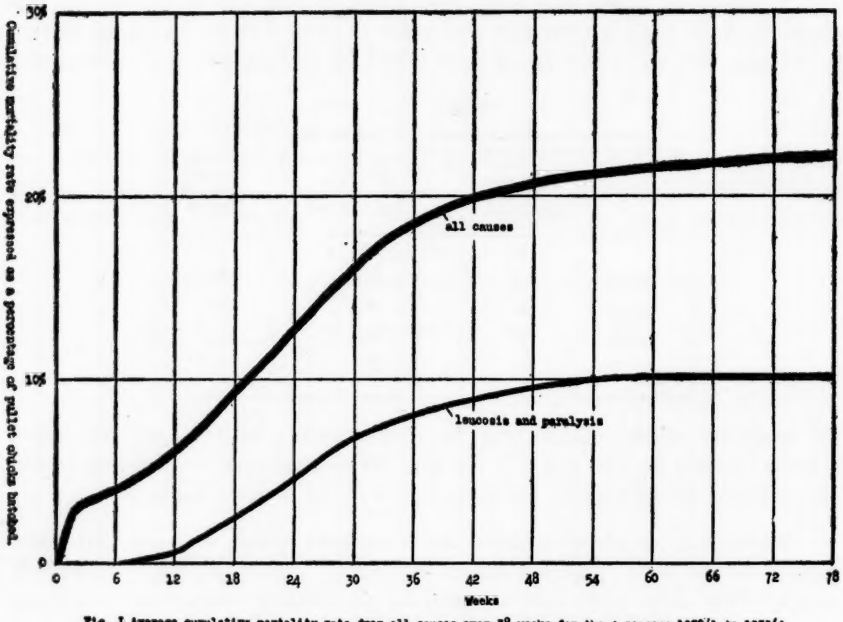

Fig. 1 Average cumulative mortality rate from all causes over 78 weeks for the 4 seasons 1950/1 to 1953/4 (the cumulative death rate from leucosis and paralysis is also shown.)

The massed figures for the four seasons show a steep rise over the first two weeks in the mortality rate which then slows down until about the eleventh week when it again begins to rise, but not as sharply as the first two weeks. This rise is associated with the onset of deaths from fowl paralysis and avian leucosis, and continues with roughly the same degree of intensity until the 45th week. At this stage until the 78th week, when recording ceased, the incidence of mortality is the lowest of the whole period. It will be noted that the incidence of paralysis and leucosis is greatly reduced from the 48th week until the end of the period.

When the four individual seasons are considered (Fig. 2) these major characteristics in the curve of the incidence of mortality are repeated. In

1950-51 with the highest death rate of 30.4 per cent, the rise in the mortality rate over the first two weeks and from the eleventh to about the 45th week is intensified. In this season the incidence of paralysis and leucosis was at its greatest. In 1951-52 the same characteristics re-appear, but there is a slowing up in the total death rate between the 30th and 35th week and also in the death rate from both paralysis and leucosis between the 28th and 39th week. The major characteristics of the death rate are repeated for 1952-53 and 1953-54, although both the total death rate and percentage of deaths attributable to paralysis and leucosis show a decline on previous years.

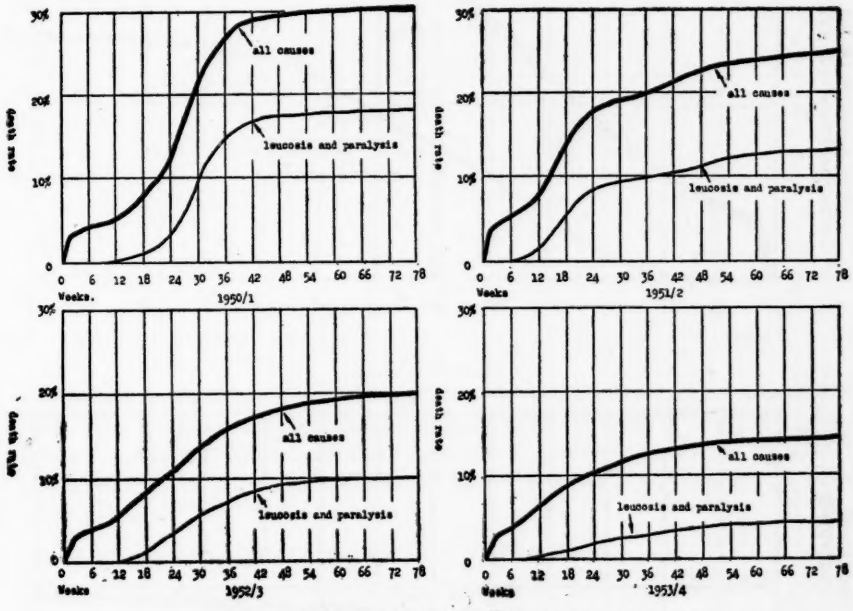

Fig. 11. Cumulative mortality rate from all causes (and from leucosis and paralysis) expressed as a percentage of chicks started each season.

Consideration of the death rates of successive hatches indicated that the incidence of mortality was generally lower in later hatches compared with early hatches, and that the percentage of deaths from paralysis and leucosis was substantially greater in early hatched birds compared with late hatched stock. The bulked figures for first and last hatches over the four seasons appear in Fig. 3. The average time lapse between the two sets of hatches is 60 days; the earlier chicks being hatched between March 1 and 3 over the four seasons and the later hatches between May 1 and 11. With the earlier hatches the same general characteristics observed for all hatches are apparent, but the slowing up in the death rate from the second to the eleventh week is not so marked. With the later hatches the incidence of mortality over the first two weeks is

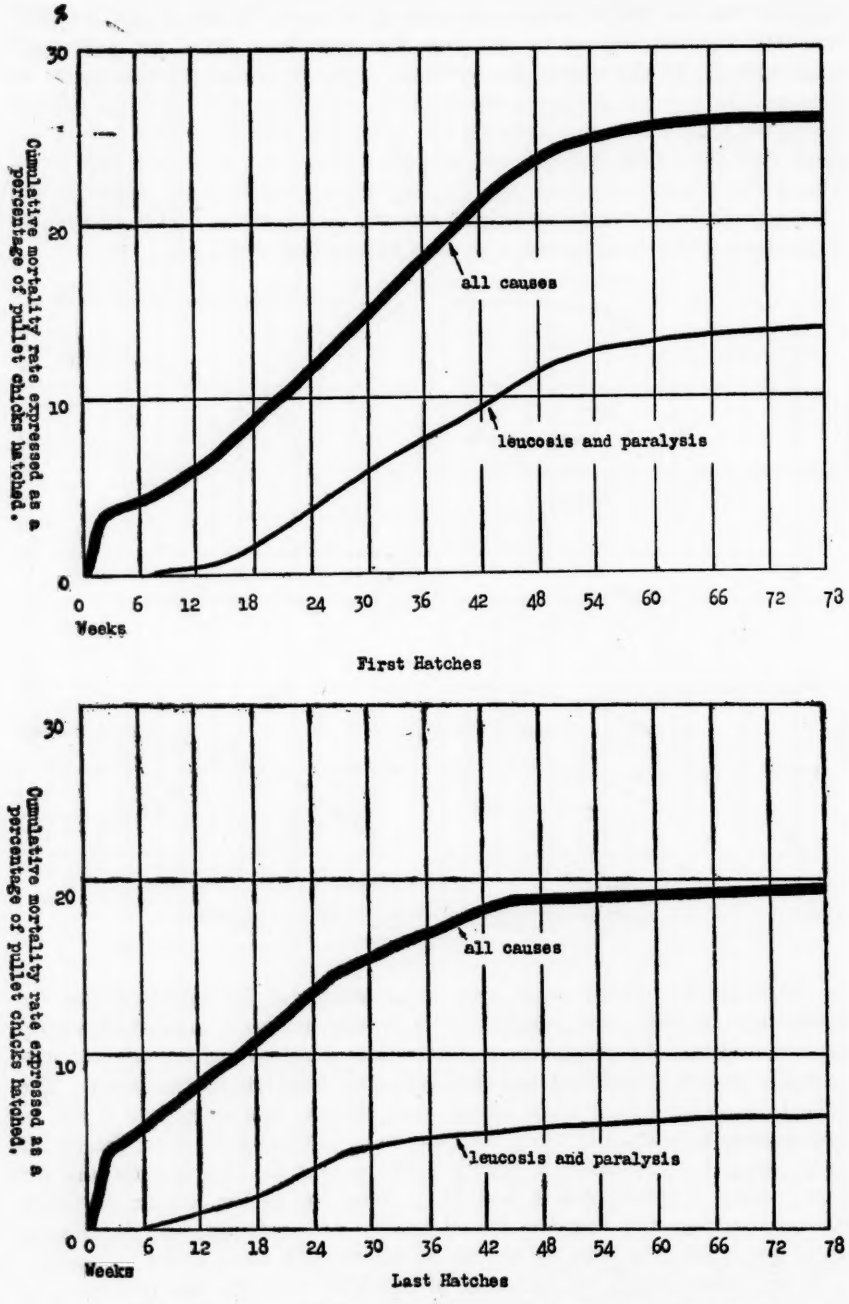

Fig. III Average cumulative mortality rate from all causes (and for leucosis and paralysis) over 78 weeks for the 4 seasons 1950-54 for first and last hatched pullets.

more emphasised, but the death rate after eleven weeks much less marked than with the earlier hatches.

Separate figures for rearing mortality and adult mortality and the incidence of paralysis and leucosis are shown in Table 4. For this purpose the termination of the rearing period is taken as 20 weeks. From this table there appears to be no correlation between rearing mortality and adult mortality. Neither does there appear any correlation between mortality during the rearing period and the incidence of paralysis and leucosis, or between adult mortality from causes other than these diseases and mortality from paralysis and leucosis. An impression of some measure of correlation between the latter groups and total

TABLE 4

Number of deaths from all causes and from paralysis and leucosis during rearing period (to 20 weeks) and over adult stage (20 to 78 weeks) shown as percentages of pullets hatched.

Season	Rearing Period			Adult Period		
	Paralysis & Leucosis	Other causes	Total	Paralysis Leucosis	Other causes	Total
	%	%	%	%	%	%
1950/51	1.1	8.0	9.1	16.3	5.0	21.3
1951/52	7.8	5.0	15.8	4.5	4.8	9.3
1952/3	1.5	6.8	8.3	7.8	4.0	11.8
1953/4	1.4	8.4	9.8	2.2	2.4	4.6
All seasons	3.3	7.8	11.1	7.0	4.0	11.0

mortality is given by a superficial inspection, but this is merely so because deaths from these diseases were responsible for the majority of cases of adult mortality. When deaths from other causes are considered separately it will be clear that no correlation exists between mortality from paralysis and leucosis and that from other causes.

(2) *The Economic Significance of the Mortality Rate.*

A record was kept of the food consumed by survivors and by birds dying. This was achieved by keeping a weekly record of food consumed and dividing it by the number of birds alive at the end of each week. It was assumed that a bird dying had up to the point of death consumed an equal quantity of food as her surviving sisters. The amount of food estimated to be consumed by such birds was deducted at the close of each week so that at the end of each 78-week period both the total amount of food consumed by the surviving birds and the amount consumed by them from day old up to the end of each successive week was available. It was possible also to calculate the amount of food consumed by any bird from day old until death supervened on the assumption that its intake of food equalled that of survivors up to the actual stage when death occurred. Clearly in the cases of a number of ailments, the birds' appetite would fail during the few days (or weeks) before death took place, and the true figure for the amount of food consumed by birds dying is no doubt a little below that attributed to them.

Birds dying prior to coming into lay represent a loss to the farmer by virtue of the cost of the chick, the amount of food consumed, and the labour and other charges expended on them. This loss in the immature bird will only be offset by any value the carcase possesses if culled prior to death. With many diseases emaciation or unattractive appearance will render the carcase value negligible, while individual birds under 10 weeks or so will have little market value even though the carcase is unaffected by the cause of death. With birds already in lay the loss sustained is reduced by the value of the eggs produced

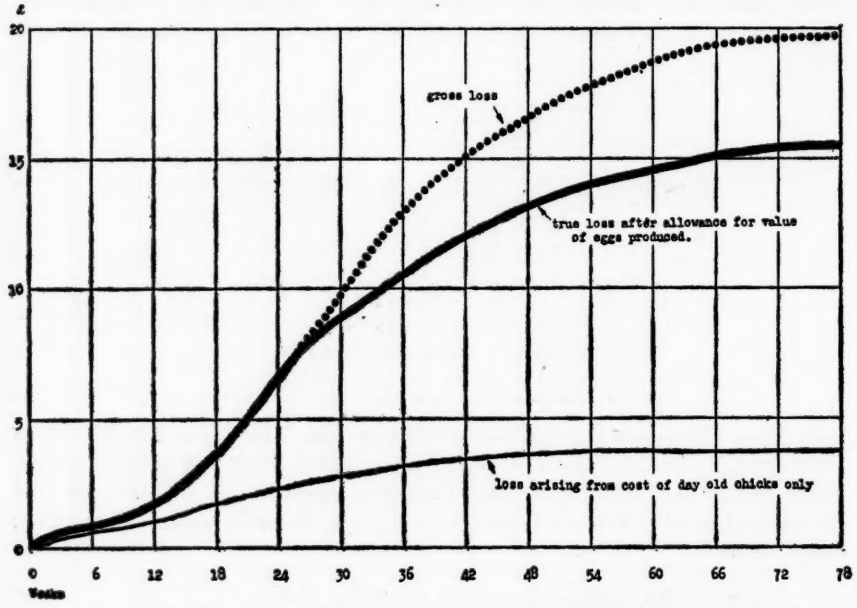

Fig. IV Cumulative true and gross financial loss per 100 pullets started resulting from cost of chick (also shown separately), food and labour based on average mortality rate of 4 seasons.

prior to the occurrence of death, while again, the carcase value will depend on the conditions already cited. In the present experiment the carcases would generally have been of little value since no culling took place; with practical poultry keepers the culling of many cases of leucosis observed in the early stages would result in saleable carcases.

It has been shown (Coles, 1954*b*) that in the total cost of purchasing, rearing and carrying a pullet over her first laying year, food amounts to an average of 65 per cent of the total costs and labour about 12 per cent. The cumulative cost of the mortality rate shown in Fig. 1 has therefore been calculated over 78 weeks on the following basis: the cost of the food consumed by birds dying estimated in the way already described at 3.25 pence per lb.; and, added to this, the cost of labour, charged as one-fifth of the food costs at the stage when death supervened. This figure is possibly very slightly above the true labour

costs but it has been employed to allow for other minor charges like overheads and depreciation. The day-old pullet chick has been charged at 3s. 4d.

The gross financial loss over 78 weeks has been calculated for the average of the four seasons on the basis of the above figures, and the result is shown in Fig. 4. In this figure, the cumulative gross loss is shown per 100 pullet chicks started, and also the cumulative loss through the costs of the day-old chicks alone. The true loss would be abated by the income from any eggs, and an attempt has been made to show how the gross loss would be reduced by the income from eggs (producer's price, 1954-5) on the assumption that up to the point of death these birds laid as well as the survivors. No attempt has been made to make allowance for the carcase value but, as many ailing birds ultimately dying would not lay, or lay below the average level over the few weeks preceding death, it is contended that the abatement for income is not unrealistic.

From Fig. 4 it will be observed that financial losses are small—mainly the cost of the day-old chick—over the first 12 weeks. From that stage onwards until about the 52nd week, the financial loss rapidly increases and would be extremely high but for the limited offsetting effect of the income from eggs from the 26th week and onwards. Beyond the 52nd week the rate of loss slows down. The period three or four months either side of sexual maturity is, therefore, the period of greatest financial loss through the incidence of mortality, but financial losses are not high through birds dying at the beginning or end of the 78-week period. It has been shown (Fig. 1) that, unfortunately, the period from the eleventh to the 45th week is the period of the greatest incidence of mortality.

It has been estimated (Coles, 1954*b*) that cost of purchasing, rearing and carrying a bird over her pullet year is approximately 60s. The gross financial loss, shown for each 100 birds on the basis described and the mortality rate experienced with this flock, amounts to £20 or 6.7 per cent of the total cost; if this gross loss is offset by the income from eggs, the net loss is 5.5 per cent. If the average death rate sustained by this flock is typical of the national flock, then with the present poultry population of the United Kingdom having an annual output of £180 million, an annual net loss through mortality of about £10 million is sustained. Furthermore, such a death rate reduces the potential egg output of the industry by approximately a further £20 million. Since this flock was managed on range, it may be contended that, as about 50 per cent of the country's adult poultry population is now housed intensively, the national mortality rate should be placed higher. There seems no real evidence for this view, and stock intensively housed from the same strains during the experimental period showed a similar mortality rate.

The rather surprising findings that earlier hatches had a higher mortality rate than later hatches prompted an examination of the egg production levels of these two groups. It is a popular view amongst poultrymen that late hatched stock (i.e., stock hatched in late April or May compared with February-March) is inferior to early hatched birds, and that this inferiority manifests itself by the presence of less vigorous chicks and poorer egg production. It has been shown

that, with this flock, the level of total mortality of the late hatched birds is below that of the earlier hatches. Mortality of the former over the rearing period was certainly a little higher, and this condition, coupled with heavy culling because of poorer production, could give rise to a belief that viability of late hatches was poor, for the egg production of the late hatches are certainly below that of early hatches.

In Table 5 is shown the average date of first egg for each season's first and last hatches together with the weighted average for the whole period. The figures for each year appear reasonably consistent. These figures are a little more favourable than the actual conditions because of the difficulty of dealing with those few birds which had not come into lay by the end of December in the year of their hatching. It will be recalled that individual egg recording ceased on December 31 in any one season. The few birds not in lay by that date were credited with first egg on January 1 following, for the alternative was

TABLE 5

Average age at production of first egg with first and last hatches (in days)

Year	Time lapse between first and last hatch	Average age of first hatch at date of first egg	Average age of last hatch at first egg	Difference between hatches
1950	50	188	240	52
1951	70	165	200	35
1952	58	181	228	47
1953	62	178	239	61
All seasons (weighted average)	60	180	221	41

to disregard them and this action would have unduly favoured the late hatches. Since the time lapse to the end of December from hatching was, of course, greater with first hatches, the arbitrary "date of first egg" for the non-laying birds in the early hatches was abated by the difference in time between the hatching dates, i.e., the period between a May 1 hatch and December 31 was 245 days, but that of a March 1 hatch 306 days; the arbitrary first egg date for the latter was therefore also included as 245 days for non-layers at December 31.

On the basis postulated, the time lapse between hatching and date of first egg for later hatches averaged 41 days longer than that of earlier hatches, and this occurred with a time lapse between the early and late hatches which averaged 60 days. The economic effect of this later date of first egg was to offset considerably the advantage of lower mortality with later hatches. The difference in the death rate between the two groups of hatches resulted in every 100 early hatched pullets sustaining losses, which in terms of chick costs, food and labour charges on the basis already described, amounted to a little over £6 more than that of later hatches. The decrease in income from egg production with the late hatched group amounted to almost £27

per 100 pullets because of the delay in date of first egg. This is based on 41 days for each bird, with a rate of lay equivalent to that of those birds already in lay, and prices at the average figure operating during the last year of this experiment. It is also customary for egg prices to be at their highest during the period when the birds are coming into lay and on these grounds the advantages of the early hatched could be still more greatly emphasised. This argument could be sustained in well-managed flocks housed under intensive conditions, but with extensive conditions and less effective management there will be a tendency for the early hatched stock to manifest a pause in production some two or three months after production has commenced and for this reason alone the higher autumn egg prices have been disregarded in assessing the potential loss.

Discussion

It may be contended that the mortality rate and the incidence of death in this flock is not representative of the average conditions in the "national flock" and in consequence the conclusions on its economic significance are not valid. However, the mortality rate in this flock over the four seasons was 22.0 per cent, and this figure approximates very closely to the figure of 21.7 per cent at the Poultry Production and Progeny Trial—this figure being the mean of the mortality rate of unculled stock from day old over 18 months for the last three years (Gordon, 1954). This trial is the only one in the country recording stock from day old to the end of the pullet year; the stock is the progeny from trial birds submitted by 100 breeders situated throughout the country and the parent birds may be taken as a fair cross-section of the stock of poultry breeders, although possibly of poultry keepers with above average stock. This latter point is one to bear in mind when comparisons are made with laying trials generally. The mortality rates over the last four years at two of the leading trials show a mean adult death rate of 10.83. This figure relates to the death rate of birds sent to the trials at about 5 to 7 months and the birds will naturally be selected from the most promising pullets. The death rate with this experimental flock amongst survivors at 20 weeks and 26 weeks respectively was 9.2 per cent and 12.4 per cent. Allowing for the range in the hatching period and, therefore, the probable age differences of birds at the trials in question, there is a good deal of similarity between the adult death rate in this experimental flock and the average adult mortality level evinced by the sample of pullets at current trials. The degree of similarity between the death rate of the rearing birds in the experimental flock and the national flock is more debatable, since no comparative figures from trials or other sources exist. The figure for young stock from the Poultry Production and Progeny trials at 6.4 per cent mortality in recent years is not strictly comparable because the numbers are reduced at random after an *average* period of 12 weeks. It has been shown that the heaviest incidence of mortality in this experimental flock invariably begins at the eleventh week. The trial's figures cited may, therefore, give a misleading impression by the fact that the numbers

are reduced at 12 weeks and the effect of the higher incidence of mortality after that date is in consequence partially masked. Nevertheless, the total death rate over 18 months of those birds retained of 21.7 per cent gives some support to the view that the rearing as well as the adult mortality in this experimental flock approximates to the average figures for the national flock.

Whether the average death rate from paralysis and avian leucosis shown in the experimental flock is representative of average conditions in the country cannot be stated with the support of any factual evidence. No doubt during the first season the death rate from these diseases was above average; during the last year it was very probably below average, owing to the success of breeding for resistance. But, if the average figure is very wide of conditions elsewhere, other flocks must have a much higher death rate from other causes since the total mortality rates show such similarity. It is therefore suggested that the mean death rate from paralysis and leucosis is not widely divergent from actual conditions throughout the country. This view is maintained in spite of the smaller number of deaths among *adult* birds, viz., approximately 25 per cent of all deaths, reported from the Ministry's laboratories in an analysis of the results of post-mortem examinations. It has, however, been argued that many birds presumably suffering from these diseases are early culled to ensure a saleable carcase or are not sent because the owner believes he knows the cause of its symptoms. This opinion, that a greater number of birds suffer from these diseases than would appear from laboratory reports, gains some credence from recent reports on the Lancashire trial already cited (Gordon, 1953). For 1951-3 "the avian leucosis complex" accounted for 41 per cent of all losses, and for a part period of 1952-4 the percentage has already reached 53. Bearing in mind the reduction in the numbers of growing stock in this trial at 12 weeks, and the manner in which the diseases manifest their greatest incidence after that date and up to about 9 months, the mean figure of 46.2 per cent with this experimental flock over 18 months seems not greatly different from that of this limited sample of the country's flocks.

It should be stated that at the present time the incidence of fowl paralysis in the United Kingdom appears to be on the increase. In contrast, avian leucosis seems to evince no proportionate increase in its incidence. The latter, during the 1940's and until recently, was relatively more common than fowl paralysis although the latter was more common before the 1940's. This experimental flock could not be regarded as typical of the *present* conditions over the country with regard to the relative incidence in the national flock of fowl paralysis and avian leucosis, but it may well be typical in that respect of the situation in a year or so's time.

If the average incidence of mortality in this experimental flock and the proportion of birds succumbing to paralysis and leucosis are accepted as typical of the country's flocks, the estimate advanced of the heavy consequent financial losses is likewise typical. The figure postulated is high and, with present-day profit margins, such an average death rate must for many individual farmers change a moderate potential profit rate to real loss or negligible profit. It is

manifest that the incidence of paralysis and leucosis is primarily responsible for a major part of the financial loss sustained, and this arises from the fact that not only are they responsible for the majority of deaths, but also from the point in the life cycle of the flock when the majority of deaths from these diseases occur. Most of them take place several weeks either side of the expected point of lay stage, when the costs of rearing are heaviest and these costs are not offset by income from eggs, or are offset to a very limited extent.

If deaths other than from leucosis and paralysis only are considered, a fairly even death rate is evinced rising very gradually after the initial sharp rise of three per cent or so mortality in the first two weeks (Fig. 5). Moreover, this death rate in both the rearing period and the laying year shows relatively little variation from year to year in spite of fairly wide variations in the inci-

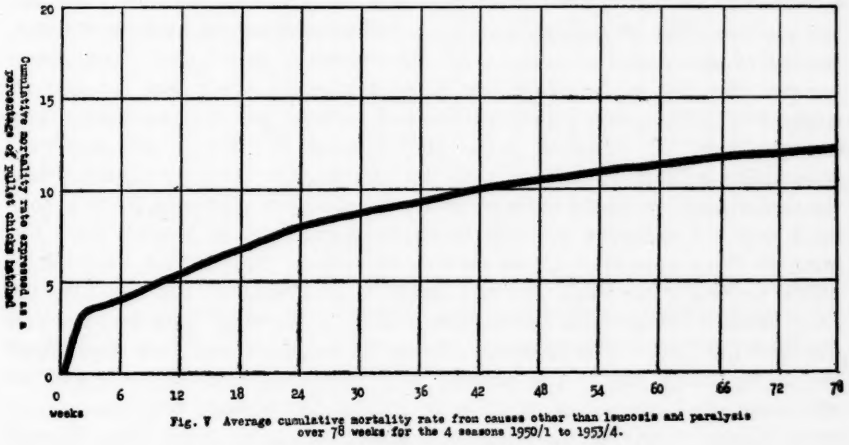

Fig. 7 Average cumulative mortality rate from causes other than leucosis and paralysis over 78 weeks for the 4 seasons 1950/1 to 1953/4.

dence of paralysis and leucosis (Table 4). The death rate to 20 weeks is mainly between 7 and 8 per cent; the adult mortality rate between 4 and 5 per cent. It is rather curious that, in this flock, the lowest mortality from other causes during the rearing period is associated with the highest death rate from paralysis and leucosis during that period. The lowest death rate in the adult period from other causes is associated with the lowest death rate from paralysis and leucosis. But the lowest death rate from other causes in the adult period follows the highest death rate from other causes in the rearing period. There is almost a kind of balancing effect between the two periods over the 78 weeks from deaths other than paralysis and leucosis in that, apart from 1950-1, they show in total little variation.

The incidence of mortality attributable to paralysis and leucosis and that from other causes gives no support for the popular opinions that there is a close correlation between total mortality and the proportion of deaths from paralysis or leucosis, and that high mortality during the rearing period is

normally followed by a high incidence of mortality from these diseases during the adult period. With regard to the first belief, it is true that a superficial impression is given that some measure of correlation exists, but this appears solely due to the fact that since paralysis and leucosis have a predominating influence on total mortality figures, the total figure follows the changes in the incidence of these diseases and the percentage of deaths from other causes is remarkably static. No relationship can be discerned between rearing mortality and deaths from paralysis or leucosis during the adult period. Nor does the analysis of mortality given in Table 2 suggest that any specific disease, such as coccidiosis, during the growing period " triggers off " paralysis or leucosis during the following adult stage.

A further popular conception which gains no support from these results is the lack of vigour attributed to later hatched chicks. It is true that deaths over the first week or so are a little higher than the percentage with early hatches, but the two series of hatches show no significant differences between the percentage of deaths due to causes other than paralysis or leucosis. The reason for the difference in the death rate of approximately 7 per cent (19 and 26 respectively) is the much higher incidence of paralysis and leucosis among early hatched birds. If, therefore, a fair interpretation of " vigour and stamina " is an ability to withstand death, then the later hatches are more vigorous than the earlier ones—or stated more precisely, the ability to withstand death is confined only to resistance towards death from paralysis or leucosis since the numbers dying from other causes show no difference. Nevertheless, the poultrymen's preference for early hatched chicks is confirmed by the fact that the early hatched chicks reach laying stage with a shorter time lapse between that date and hatching. The increased egg production so secured more than offsets the higher death rate. The possible relationship between fecundity and an above-average incidence of paralysis or leucosis has already been commented upon (Coles, 1955) as the highest incidence of death from these diseases invariably occurs with birds which have just commenced to lay. It has been the experience of the author that the most promising looking groups of sisters, those appearing well grown and vigorous above average, are those among which paralysis and leucosis most rapidly and extensively manifest themselves when these diseases are widespread throughout the flock. This experience seems not uncommon with commercial poultry-keepers. It may, therefore, be that, if any relationship does exist between fecundity and a tendency to manifest these diseases, the death rate would in consequence be higher among those birds laying at a higher level and at an earlier date than their sisters. This opinion suggests that if later hatched pullets were subjected to intensive housing and lighting in an endeavour to promote earlier laying, this end might be secured at the expense of an increase in the death rate from paralysis and avian leucosis. On economic grounds, however, such an action with the possible dual consequence indicated would be justified. A point of some interest is that culling, if practised in this flock, would not have removed many of those birds likely to die, and so would not have prevented the cost of rearing them.

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A comparison of the mortality rate of the hen-bred and pullet-bred birds only partially bore out another widely accepted view that hen-bred stock is more viable and more resistant to paralysis (Table 6). Total average mortality was approximately the same over the four seasons. The failure of the hen-bred stock to show its expected superiority may, however, be attributed to the endeavours to establish resistance to paralysis and leucosis. Elsewhere the author has shown that, in the early stages of such work, dams with paralysis-free daughters from their pullet year mating appear to suffer a severe breakdown in their hen-bred progeny when resistance has not been well established and

TABLE 6

Incidence of mortality (from paralysis and leucosis and other causes) in hen-bred and pullet-bred birds

Season	Hen-bred			Pullet-bred		
	Paralysis and Leucosis	Other causes	Total	Paralysis and Leucosis	Other causes	Total
	%	%	%	%	%	%
1950/51	11.1	11.1	22.2	17.9	13.1	31.0
1951/52	17.9	9.4	27.3	10.0	12.3	24.3
1952/53	15.7	3.6	19.3	8.7	11.4	20.1
1953/54	2.1	7.2	9.3	3.8	11.4	15.2
All seasons (weighted average)	13.6	8.3	21.9	9.6	12.5	22.1

exposure is severe (Coles, 1955). Towards the end of the experiment the hen-bred progeny manifested the expected greater viability, and throughout the deaths from causes other than paralysis and leucosis were below the percentage shown by the pullet-bred stock.

Summary and Conclusions

An account is given of the incidence of mortality in a flock of White Leghorns over four successive seasons, each of 78 weeks' duration. The importance of the stage in the 78-week period when death supervenes is discussed, and an attempt made to demonstrate the extent of the financial losses incurred by the poultry industry through a mortality rate consistent with that found with this flock, and with deaths occurring at the stages found. It is argued that both approximate to the average found in the national flock. On the basis of current costs and the present-day size of the poultry industry, the actual loss and loss of potential income is estimated at about 30 million pounds annually.

Attention is drawn to the important role of paralysis and leucosis in controlling the death rate, although their incidence is not apparently influenced in any way by the number of deaths from other causes during rearing or in the adult stage. Neither is the percentage of deaths from other causes influenced by the incidence of deaths from paralysis or leucosis. The importance of these diseases on economic grounds arises not only from the total number of birds

dying from these diseases—about 50 per cent of all deaths—but also from the stage at which the birds die, i.e., around maturity, when the heavy expenditure on rearing is not offset by much in the way of income from egg production. Hen-bred pullets generally showed a lower level of mortality from diseases other than paralysis and leucosis over the whole period when compared with their pullet-bred sisters, and a lower incidence of death from paralysis and leucosis towards the termination of the experiment. At the beginning of the period, however, when only a slight degree of resistance in the flock was established, the death rate from these diseases was greater in hen-bred pullets than in their pullet-bred sisters.

First hatches showed the same death rate from causes other than paralysis and leucosis as hatches taken off approximately 60 days later. But the former evinced double the death rate from paralysis and leucosis. Since the later hatched birds produced their first egg approximately 41 days later from date of hatching than the early hatches, the financial return from late hatched stock is substantially lower than from the former. The possibility of a connection between the higher death rate of the more prolific early hatches and the higher incidence of paralysis is suggested.

Acknowledgment

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REFERENCES

Campbell, J. G. (1954): *Xth World's Poult. Cong., Edinburgh, 2,* 193.
Coles, R. (1954): *Poult. Sci., 33,* 350.
Coles, R. (1954*b*): *J. Royal Ag. Soc. Eng., 119* (in press).
Coles, R. (1955): *Poult. Sci. 34* (in press).
Dudley, F. J., Dobson, N., and Gordon, R. F. (1941): *Vet. Record, 53,* 323.
Gordon, R. F. (1953): *Proc. XVIth Int. Vet. Cong. Stockholm, 2,* 1054.
Gordon, R. F. (1954): *Vet. Record, 66,* 828.
Kennard, D. C. (1933): *Poult. Sci., 12,* 335.
Waters, N. F. (1951): *Poult. Sci., 30,* 531.

POST-PARTURIENT METABOLIC DISORDERS IN THE COW

An approach to therapy with special reference to prolonged recumbency

By E. F. LEWIS

Department of Medicine, Royal Veterinary College, Streatley

THE problem of the cow which is unable to rise to its feet following parturition and the so-called metabolic disorders associated with that period is well known to the veterinary practitioner in agricultural areas. In past years the incidence appeared to be low but a marked increase is now being reported by some veterinary surgeons in the southern counties. Only a careful survey could substantiate this and a good deal of biochemical research would be required to establish a satisfactory explanation but it is felt that a brief discussion of some of the common findings associated with prolonged recumbency might be of value in focusing attention on a problem which could become economically serious.

The history of milk fever points beyond question to a waxing incidence in direct proportion to the increasing use of intensive farming methods. To some degree the second world war provided a check by reducing the feeding stuffs available, but research was given momentum by the exigencies of the national emergency. The application of the many theories put forward by agronomists in the immediate post-war period as a result of this research is now bearing fruit. The great variations in farming practice which aim at getting the utmost out of the long-suffering animal make it necessary for the veterinary surgeon to keep his ideas on normal standards constantly under review. The dietary requirements of the modern dairy cow in connection with the assimilation and storage in reserve of the common mineral elements must surely vary with the changing systems of pasture management, food preparation and, indeed, with the changing nature of the foods themselves. Full recognition of this fact and its implications must assist, therefore, the appreciation of any disorder which may arise as a clinical deviation from the accepted common syndrome.

It has been postulated, and quite rightly, by eminent research workers in this field, that metabolic disorders in principle are exaggerated effects of those changes which occur normally in every animal. The recent experimental work of Moodie, Marr and Robertson (1955*a*) supports the hypothesis of Allcroft (1947) in this respect. The speed and volume of these changes are linked with the ability of the animal to mobilise requirements from reserves and to utilise available supplies of necessary chemical substances in the food or therapeutic agents. This ability is governed by the over-all pattern of farming practice, but the recovery of the patient, whether it be spontaneous or in response to

therapy, is related to the extent of chemical changes in the animal's tissues. Beyond a certain limit pathological changes occur which are not immediately reversible. The first factor, therefore, in overcoming the possibility of prolonged recumbency is the speed with which counter measures are implemented. As a general rule they cannot be applied too quickly. Further factors, however, which influence directly or indirectly the alacrity of the response to therapy are the chemical compounds used, their dosages and the routes whereby they are administered.

The ratios existing between the blood chemical elements are of greater importance than the consideration of any individual element and it is the marked disturbance of these balances which leads to one or other of the clinical syndromes associated with metabolic disorders. To produce the most satisfactory response to treatment it is necessary to know the manner and degree in which these ratios have been disturbed, but it is not usually considered practicable to couple therapy directly with blood analysis. The information gained by systematic sampling of cases in an area, however, would lead the practitioner to better appreciation of the clinical condition with which he is dealing.

It is impossible to state a general formula which lays the responsibility for prolonged recumbency at the door of any particular element, but ever since Fish (1929, 1930) demonstrated the nature of blood phosphate changes in milk fever cases, evidence suggests that this element is at least partly concerned in the exacerbation of general symptoms. The type of case which shows one or more relapses or which fails to respond completely to initial treatment draws attention to the phosphate side of Ca/P ratio. As confirmed recently by Moodie and his colleagues (1955*b*), the blood phosphate level is closely allied to calcium in its fall and rise. This lends colour to the suggestion that the concurrent use of phosphates with calcium salts is the rational routine therapeutic approach. The inclusion of magnesium on the other hand should not be regarded as a routine measure. The work of Seekles, Sjollema and Van der Kaay (1932) and Allcroft and Godden (1934) associates a state of hypermagnesæmia with normal parturition. Messervy (1948) linked the comatose stage of milk fever with marked hypermagnesæmia and Moodie and his colleagues (1955*c*) showed that the serum magnesium levels tended to increase reaching their highest point about one day after normal calving. The inclusion of magnesium, therefore, might well retard the rapid return to normal in the animal affected by clinical hypocalcæmia.

A full dose of at least 3 oz. of calcium borogluconate should be administered initially. This figure applies to the smaller breeds but may well reach 6 to 7 oz. for the larger animals. The tendency to underdose is responsible for incomplete response and predisposes to prolonged recumbency. Various proprietary brands of Injecto : Calcii : Borogluconatis are marketed and users of these products should take care to think in terms of the actual quantity of calcium borogluconate being administered. Some of these products contain phosphates which are compatible with calcium solutions but as a separate source, sodium

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acid phosphate in 1 oz. doses may be injected intravenously and followed up by a similar daily dose given by mouth. The requirements of the Channel Island breeds of dairy cattle and in particular the Jersey, seem to be proportionately higher than those of other brands.

The route of administration is also a matter for careful consideration. Acute hypocalcæmia should be countered by suitable drugs administered the quickest way, i.e., by intravenous injection. Solutions of calcium borogluconate, up to 15 per cent in strength, are best given by means of a gravity apparatus, not less than five minutes being taken over the operation. The use of concurrent phosphates has already been mentioned and it should be reiterated that the use of magnesium salts should be entertained only in those cases where clinical findings of tetany, convulsions or other features indicate a state of hypomagnesæmia.

Whether or not a second dose should be given by subcutaneous injection to simulate depot therapy in preventing relapse is determined by the general and clinical history of the case, but there is a risk that such a practice might in some areas mask a relapse in the form of a subclinical condition. The omission of the initial intravenous injection in favour of the subcutaneous route may, however, be accompanied by delays of anything up to twelve or more hours before the animal can be regarded as normal. It is in this delay that some of the factors responsible for complications are likely to arise.

The following figures apply to a number of cases of clinical "milk-fever" encountered during the period September, 1942, to September, 1948. The routine method of therapy was the intravenous injection of calcium borogluconate, solutions being prepared immediately before use in the doses outlined above.

			Approx.
	Total number of cases	755	%
Group 1.	Number responding to initial treatment ...	337	45.0
Group 2.	Number given subsequent treatment ...	225	30.0
Group 3.	Number exhibiting clinical relapse ...	193	25.0
	Number showing prolonged recumbency	12	1.6

The animals included in Group 2 exhibited some abnormality after initial treatment. The signs varied considerably on different farms, in some instances the patient's refusal of food, usually a bran mash offered some twelve hours later being regarded as early evidence of the presence or return of the disorder. A successful response to treatment was in most cases confirmed by the return to the animal of a desire to drink. It would be impossible, however, to draw a definite line between Group 2 and the group showing relapses evinced by the exhibition of definite clinical features on one or more occasions after the initial attack.

The proportion of relapsing cases to those requiring subsequent treatment may be regarded as being very high, but it should be remembered that con-

current use of phosphates had not been practised and it is probable that a number of the 225 animals given subsequent treatment may have responded more slowly without secondary attention. In spite of this, the number exhibiting prolonged recumbency was low, the group being limited to those animals which failed to rise within twelve hours of the commencement of treatment, and included six animals which responded at about the 24-hour period, one animal which remained recumbent for five days, three animals which did not get up and were subsequently destroyed, and two animals which died after 12 to 24 hours as a result of hepatic dysfunction and toxæmia of chemical origin.

In the later stages of the same period a small number of animals were treated with calcium borogluconate and sodium acid phosphate, both drugs being given on the occasion of the first visit. The twenty-two cases receiving this therapy showed a significant decrease in the percentage of relapses and in the number requiring supplementary treatment. It is considered, however, that the comparison of these few cases with the previous 755 offers no proper basis for discussion and details, therefore, are omitted.

Exaggerated and prolonged effects of blood mineral imbalance lead to a second group of factors usually associated with extreme degrees of abnormal recumbency. It is in this group that pathological changes have occurred which are only resolved by the natural processes of tissue repair or may in fact be so extensive that natural repair is impossible. Degenerative changes in muscles, tendons and similar tissues have long been recognised as complications and are most often associated with the hind limbs and the lumbo-sacral regions. They may be slight and very local, e.g., cloudy swelling of muscle fibres, requiring the minimum time for repair, or marked and developed beyond possible resolution. Of the latter type the degenerative changes seen in the gastrocnemius muscle of the Jersey, especially at the insertion of the tendon into the muscle and leading in some cases to spontaneous rupture, might be cited as an extreme example. In all these cases the blood chemical picture can be restored by suitable therapy but the animal remains recumbent or unable to stand until sufficient repair of damaged tissues has taken place. It is possible to visualise many changes associated with wide permutations of the clinical picture which are directly connected with the syndrome. Patience and skilled nursing are essential in dealing with any of those conditions where the prognosis is not hopeless from the outset.

Associated indirectly with the mineral changes are all those factors which usually arise accidentally. Included in this group are the luxations, sub-luxations, fractures, strains, sprains and rupture of ligaments. These usually arise through inco-ordinated action of the locomotor system in the early or late phases of the paretic stage. Quite often they arise because of slowness in the response to treatment and bring a number of surgical problems in their wake.

Nervous activity in those nerves supplying the powerful hind limb muscles may be impaired or inhibited directly by the mineral changes and indirectly by trauma sustained during the passage of the fœtus through the pelvis or by

prolonged pressure from the gravid uterus in those cases where clinical hypocalcæmia affects the animal prior to calving. Over distension of the urinary bladder which may occur as a result of the original paresis or paralysis sometimes persists, and unless relieved by catheterisation may by pressure on pelvic nerves, cause continued recumbency.

A varying degree of ketosis inevitably accompanies the original disorder and in itself may be responsible for prolonged recumbency by virtue of the toxæmia of chemical origin. This systemic reaction which removes from the animal the desire or physical ability to stand, requires specialised treatment along one of the modern recommended lines. Impairment of liver function in ketosis is associated with fatty changes in the liver parenchyma to the detriment of other aspects of hepatic function and the state of toxæmia progresses rapidly. These cases show a temporary improvement following simple calcium therapy but usually relapse in one or two hours to the original semi-comatose state. It is important also to differentiate the clinical milk fever syndrome with its attendant complicating features from the superficially similar picture given by any advanced toxæmia or septicæmia of biological origin. Included in this last category but falling outside the scope of the paper are the conditions occurring during the nursing of a cow which is recumbent for four or more days. Open joints resulting in septic arthritis, pressure sores, mastitis, metritis and pulmonary infections are all well-known complications and are usually accorded a most unfavourable prognosis.

Summary

An attempt has been made to discuss the parts played by animal husbandry and nutrition, in influencing the incidence and nature of parturient metabolic disorders with special reference to the complicating factor of prolonged recumbency.

The therapeutic agents, their dosages and methods of administration are discussed with emphasis on their effects in reducing the incidence of prolonged recumbency.

Other factors which may be responsible for this complicating phenomenon are also briefly mentioned.

REFERENCES

Allcroft, W. M., and Godden, W. (1934): *Biochem. J., 28,* 1004.
Allcroft, W. M. (1947): *Vet. J., 103,* 2.
Fish, P. A. (1929): *Cornell Vet., 19,* 147.
Fish, P. A. (1930): *11th Int. Vet. Congr., London, 2,* 330.
Messervy, A. (1948): Rep. Proc. Conf. on Grassland and Animal Health, 1948, 94.
Moodie, E. W., Marr, A., and Robertson, A. (1955*a*): *J. comp. Path., 65,* 20-36.
Moodie, E. W., Marr, A., and Robertson, A. (1955*b*): *Ibid.*
Moodie, E. W., Marr, A., and Robertson, A. (1955*c*): *Ibid.*

PLAQUE-LIKE PLACENTAL LESIONS IN CATTLE

By P. G. MILLAR

Ministry of Agriculture, Fisheries and Food, Weybridge

In 1948, we noticed well-defined lesions on the fœtal membranes of a cow in the Islington abattoir. Being aware of the possible existence of infective factors, other than those already appreciated in the problem of bovine infertility, the appearance of these rather striking lesions aroused great interest. During the period to June, 1952, we had the opportunity to obtain a further nine specimens. Five of these were from abattoirs, and four were obtained from cases of abortion. It was not possible to determine the clinical history of the others. In three of the instances of which we had knowledge, the animals were destroyed because of emaciation of undetermined cause, Johne's disease and tuberculosis being absent. The possible significance of these well-defined lesions emphasised the necessity to study their nature by laboratory techniques.

Description of Lesions

Flattened, plaque-like, symmetrical masses of polyhedral cells are present on the chorionic membrane and adherent to it by a central node. The fixation is weak, especially in "advanced" cases. The usual features of inflammatory change are absent. The lesions found in early gestation are small (0.1 to 0.2 c.m.) and are distributed in an area around the hilus of the umbilical cord. As gestation proceeds the plaques increase in size, tend to coalesce and to involve a greater area of the chorionic membrane.

In no instance was it possible to demonstrate the presence of brucellosis, *Vibrio fetus* infection, trichomoniasis, tuberculosis or Johne's disease. Fresh suspensions of the lesions proved to be bacteria-free when cultured aerobically and anærobically. Because these findings suggested the possibility that a virus might be responsible for the onset of these lesions and for the gestation failure, the following experiments were made.

Initially attempts were made to reproduce the condition in laboratory animals and to demonstrate the effects of inoculations on them.

A. Guinea-pigs.

I.—Inocula of bovine origin: (a) Emulsions of suspected lesions; (1) Untreated, (2) Diluted, (3) Filtered and diluted, (4) Diluted and treated with penicillin and streptomycin; (b) Placental fluids; (c) Emulsion of placental membrane and lesions.

Thirty-two pregnant and thirty-one non-pregnant guinea-pigs were inoculated intraperitoneally or intravaginally.

Observations

The mortality rate within a few days after inoculation was low, as also was the abortion rate. The percentage of dead and non-viable fœtuses was similar to that experienced among the general guinea-pig breeding stock.

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PLATE I

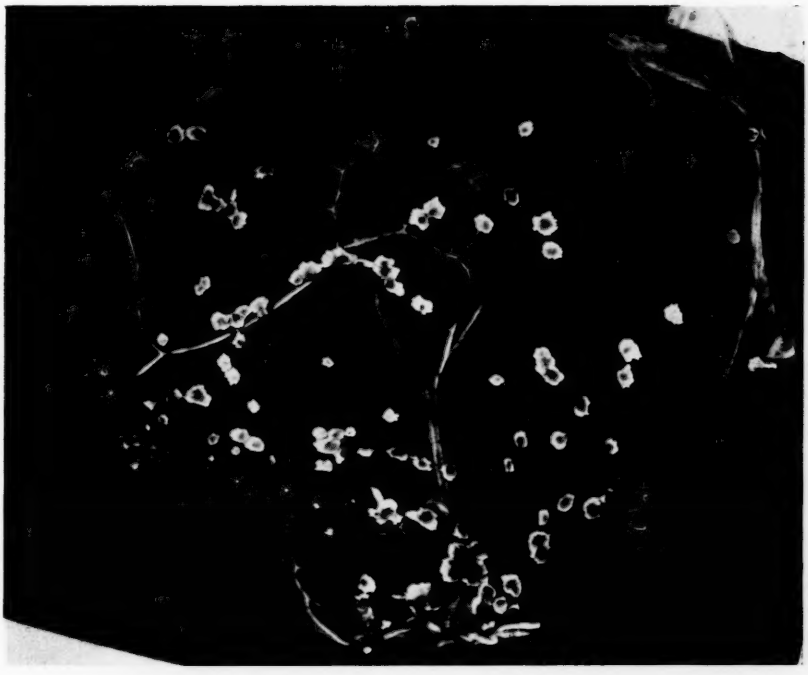

Fig. 1

"Seed" plaques at 10th week of gestation.

(*Article by Millar, page* 258)

XUM

PLATE II

Fig. 2

Placental plaques at 8th month of gestation.

(*Article by Millar, page 258*)

II.—Guinea-pig/guinea-pig passage: From guinea-pigs which had been inoculated, materials were collected for the inoculation of other guinea-pigs. These included emulsified uteri of adults and fœtuses, livers of adults and young and filtered uterine fluid. These further passages produced insignificant reactions similar to those elicited by the original inoculum.

B. Goats.

Two pregnant goats were inoculated intraperitoneally at an advanced stage of pregnancy with a suspension of bovine lesions. Neither thermal nor clinical reactions were observed after inoculation. Both animals kidded normally and no abnormality has been seen since parturition. It was not possible to demonstrate the presence of anti-bodies in the sera of these goats. Further, four non-pregnant goats were inoculated intravaginally with a suspension of bovine lesions. The inoculation was not followed by thermal or clinical reactions. Serological techniques failed to reveal the presence of antibodies in the sera collected from these animals. One goat was destroyed at 6-weeks after inoculation and a post-mortem examination failed to reveal the presence of any abnormality.

C. Cattle.

Two heifers were inoculated intravaginally with suspensions of bovine lesions and showed no thermal reaction. The genital organs remained apparently normal. Within a few days, both heifers were inseminated and became pregnant, ultimately calving without evidence of abnormality.

D. Embryonated Hen-eggs.

Two experiments were made, employing the following inocula: (1) Seitz filtrate of lesions, (2) Fœtal membrane suspensions, treated with penicillin and streptomycin.

Ten-day embryonated hen-eggs were used, and no mortality of embryos occurred. Emulsified C.A.M. from these eggs, inoculated into two heifers, produced neither thermal nor clinical reactions. Both were subsequently inseminated and conceived. Four guinea-pigs (non-pregnant) were similarly inoculated. As no clinical reaction took place, all were killed after 38 days. Post-mortem examination revealed no abnormalities.

Serological Investigation

The serological investigation was handicapped to some extent, by the lack of other body-tissues and fluids from naturally affected animals. However, serum samples collected from the experimental cows and goats, before and after inoculation, did not show any antigen-antibody reaction.

The antigens used had been prepared from the following: (1) Emulsions of affected fœtal membrane, (2) Emulsions of lesions, (3) Guinea-pig liver emulsions, (4) C.A.M. emulsions from eggs.

Discussion

These experiments failed to reveal the presence of an infective agent and suggest that the plaque-like lesions are not of extrinsic significance. Although their striking appearance is such as to suggest that they might be transmitted within a herd and be associated with lowered fertility, the available evidence is that they are of non-infective significance. We have felt that the characteristics of these lesions are such as to attract the attention of anyone interested in the problems of reproduction in cattle and that they warranted investigation.

Acknowledgment

I would like to thank Dr. Mansi for his advice on the serological aspects of this investigation.

THE STUDY OF INORGANIC PHOSPHORUS IN BLOOD IN EGYPTIAN BUFFALOES, NATIVE AND CROSS BREED CATTLE

By HASSAN SALEM, Ph.D.(London), and
HUSSIEN REDA, M.V.Sc.(Cairo)
Faculty of Agriculture, Cairo University

In the early work of Hamersa (1937), it was shown that the mean value for bovine serum inorg. P (subsequently referred to as IP), was about 7.0 mg. per 100 c.c. Sabovljev (1938) found in normal serum cattle, a mean of 9.66 mg. per cent. It has been stated by Jones (1943), that the blood IP tended to be from 4.5 to 7.1 mg. per cent for tropical cattle, while Dukes (1943) found that serum IP ranged from 2.3 to 9.6 mg. per cent for temperate cattle. Payne and Clark (1946) observed higher serum IP in bulls than in cows. Their figures showing about 7.3 mg. per cent for yearling bulls and 4.7 mg. per cent for older herd bulls. Fourie and Deacon (1947) found that the blood IP content of cattle of both sexes gradually diminishes as the animals grow older, ranging from about 6.0 mg. per cent in calves to about 4.0 mg. per cent in the adults, although Terrie, *et al.* (1946) could detect no variation with age. Bjoberg (1939) stated that the IP in the blood fell sharply at parturition, even to as low as one-third of the normal value, whilst Allcroft and Godden (1934) showed only a slight decrease in the blood IP of cows immediately before and at parturition and Kennedy *et al.* (1939) reported the IP of bovine blood to be little affected by gestation. Mullik and Pal (1944) also reported that

there was no significant variation in the IP of the blood during lactation or pregnancy.

During the course of an investigation in this laboratory into the different changes occurring in the IP constituents, it was considered desirable to endeavour to study the changes in the IP in the blood of Egyptian cattle and buffaloes under different physiological conditions.

Material and Technique

The work was carried out on two herds of animals belonging to the Faculty of Agriculture, 30 buffaloes and 35 cattle (22 Egyptian Balady and 13 cross-

TABLE I

MEAN AVERAGE INORGANIC PHOSPHORUS LEVEL IN BUFFALOES AND CATTLE

No. of Animals	Sex		IP level mg. %. Max	Min	Aver.
4	Males	3-9 years	9.0 ± .3	6.2 ± .7	7.1 ± .5
12	Females	3-8 years	7.0 ± .4	4.4 ± .3	7.1 ± .5
9	Heifers	4-18 mths.	9.0 + .2	8.0 ± .3	8.6 ± .5
5	Calves	1-16 weeks	8.6 + .3	7.3 ± .5	8.2 ± .3
		Egyptian Cattle			
5	Bulls	3-7 years	8.0 ± .4	5.8 ± .3	6.6 ± .3
8	Cows	4-10 years	6.3 ± .5	4.0 ± .8	4.5 ± .2
4	Heifers	12-18 mths	7.7 ± .3	7.4 ± .3	7.6 ± .4
3	Calves	1-12 weeks	8.0 ± .3	7.6 + .5	7.8 + .2
		Cross Breed			
2	Bulls	4-6 years	8.0 ± .4	6.1 ± .2	6.4 ± .3
7	Cows	3-9 years	5.6 ± .5	4.1 ± .4	4.3 ± .5
3	Heifers	12-14 mths.	7.6 ± .3	6.8 ± .3	7.2 ± .2
1	Calves	1 week	9.8	7.2	8.0

breed, shorthorn). There is only one breed of Egyptian cattle, the Balady. The experimental animals were males, non-pregnant and pregnant females. Estimations of IP in the blood of pregnant animals were made throughout the whole period of gestation. All animals used in the experiments were healthy and were fed a well-balanced diet.

Analytical Method.

The blood samples were taken from the jugular vein into clean tubes after discarding the first drops.

The method used was adapted from that of Fiske and Sabbarow (1925), the protein of the blood being precipitated with trichloroacetic acid, the protein free filtrate treated with an acid molybdate solution, and the phosphomolybdic

acid reduced by 1.2.4. aminonaphtholsulphonic reagent. The density of the blue colour produced was determined in the photometer, using a 660 mμ filter.

Results are shown in Tables I and II.

Discussion and Summary

In general. buffaloes gave higher IP blood level (7.1 mg. per cent males) than either Egyptian or cross-bred cattle. The authors' results for normal

TABLE II

MEAN AVERAGE INORGANIC PHOSPHORUS LEVEL DURING PREGNANCY AND AFTER PARTURITION

IP Level mg. per 100 ml. Serum

(Pregnancy) Months									Weeks Post-partum				
2	3	4	5	6	7	8	9	10	Part.	1	2	3	4
Buffaloes													
	5.5 ±.6	5.5 ±.4	5.6 ±.5	5.7 ±.5	5.9 ±.4	6.0 ±.2	6.2 ±.6	4.5 ±.5	4.8 ±.3	5.3 ±.4	5.4 ±.8	5.5 ±.3	5.5 ±.5
Egyptian Cattle													
5.0 ±.3	5.2 ±.6	5.1 ±.4	5.2 ±.5	5.4 ±.2	5.5 ±.5	5.7 ±.2	5.9 ±.5		4.5 ±.7	4.8 ±.6	5.0 ±.3	5.0 ±.5	5.0 ±.4
Cross Breed													
4.8 ±.4	4.9 ±.4	4.9 ±.7	5.0 ±.6	5.1 ±.6	5.3 ±.5	5.5 ±.3	5.7 ±.2		4.3 ±.3	4.6 ±.2	4.7 ±.5	4.8 ±.3	4.8 ±.4

Egyptian cattle (6.6 mg. per cent males) agree with those of Hamersa (1937). The level of the serum IP of cross-bred cattle (6.4 mg. per cent males) was nearly the same as that of the Egyptian cattle, in agreement with the findings of Jones (1943) and Dukes (1943).

In all animals examined, males gave a higher level than females, as reported by Payne and Clark (1946). Age had an influence on the IP level as found by Payne and Clark (1946) and Fourie and Deacon (1947).

It is quite obvious from Tables I and II that there were significant age and sex differences in both buffaloes and cattle.

During pregnancy the IP was little affected by gestation, beginning close to the average level and increasing slowly up to very late pregnancy. After parturition the value fell slightly below normal and then gradually rose again. These results agreed with those of Kennedy, *et al.* (1939).

One of the male Egyptian cattle gave about 3.2 mg. per cent of IP, which was about half the normal amount.

REFERENCES

Allcroft, W. M., and Godden, W. (1934): "Changes in Ca. and Mg. of Serum and in the IP of the Blood of Cows." *J. Biochem., 28,* 1004.

Dukes, H. H. (1943): "The Physiology of Domestic Animals." New York: T. B. Comstock.

Fiske, C., and Subbarow, V. (1925): "Determination of IP." *J. Biol. Chem., 66,* 375.

Fourie, J. M., and Deacon, E. A. (1947): "IP in the Blood of Cattle in Relation to Age." *J.S. Afri. med. Ass., 18,* 101.
Hamersma, P. J. (1938): "Chemical Blood Studies." *Vet. Bull., Dec., 8,* 811.
Jones, E. R. (1943): "The Effect of Tropical Environment on the Blood and Milk of Cattle." *Vet. Rec., 55,* 128. (Quoted in Bull. No. 9, The Hannah Dairy Res. Inst., 1950, p. 54.)
Kennedy, W. L., Anderson, A. K., and Shijley, J. F. (1939): "Studies on the Composition of Bovine Blood." *J. Dairy Sci., 22,* 251.
Mullik, D. N., and Pal, A K. (1944): "Studies on the Composition of the Blood of Farm Animals." *Indian J. Vet. Sci., 13,* 146.
Payne, M. G., Clark, G., and Kinjman, H. F. (1946): "Blood Level of Ca. and IP in Hereford Cattle." *J. agric. Res., 72,* 357.
Sabovljev. A. (1939): "The Blood Serum Ca. and IP in Domestic Animals." *Vet. Bull, 9,* 582.
Sjoberg, K. (1941): "Changes in the IP of the Blood of Cattle at Parturition." *Vet. Bull., 2,* 264.
Terrie, A. E., Kenner, H. A., and Marrow, K. S. (1946): "Studies on the Chemical Composition of Calf Blood." *J. Dairy Sci., 29,* 663.

ABSTRACTS OF LITERATURE

FIELD TRIALS ON THE PREVENTION OF BODY-STRIKE IN SHEEP BY THE USE OF D.D.T. AND B.H.C., by I. L. Johnstone and Marion T. Scott. *The Australian Veterinary Journal,* Vol. 27, No. 4, pp. 79-82.

TRIALS reported in this paper show that the application of D.D.T. or B.H.C. is an effective and practical control measure under field conditions for the prevention of body-strike in sheep. A concentration of 1 per cent D.D.T. or 0.1 per cent gamma isomer B.H.C. applied at the rate of approximately 2 pints per sheep was effective, although in one trial 1 per cent D.D.T. at the rate of 1 pint per animal also seemed to be effective. Application was by a jetting plant or by the top set of nozzles in a power-spray unit. Protection was considered to last at least a month and, under mild fly-wave conditions, it lasted 10 weeks.

THE OCCURRENCE OF *Ascaris vitulorum* GOEZE, 1782, IN CALVES IN AUSTRALIA, by R. K. Keith. *The Australian Veterinary Journal,* Vol. 27, No. 6, p. 129.

KEITH records the occurrence of *Ascaris vitulorum* in calves in the Armidale district of New South Wales, this being the first occasion on which the parasite has been reported from Australian cattle. The examination of fæcal samples from two- to eight-week-old calves showed the presence of eggs in four cases. The life cycle of the parasite is discussed, especially in respect to the occurrence of pre-natal infestation.

SUSPECTED TETRACHLORETHYLENE POISONING OF SHEEP, by W. H. Southcott. *The Australian Veterinary Journal,* Vol. 27, No. 6, pp. 130-131.

SOUTHCOTT records deaths amongst young Merino sheep in winter or early spring during 1945, 1948 and 1949. The drenching was with 4 c.c. of tetrachlorethylene in 4 c.c. mineral oil immediately preceded by 15 c.c. of a 5 per cent copper sulphate solution in 1945, with 4 c.c. of the pure drug preceded by 20 c.c. of 5 per cent copper sulphate solution in 1948, and 5 c.c. of the pure drug preceded by 20 c.c. of 5 per cent copper sulphate solution in 1949. The post-mortem appearances resembled those of carbon tetrachloride poisoning, and it is suggested that under certain undefined circumstances tetrachlorethylene

may be toxic to sheep, similarly to the former drug. It is recommended that the necessary precautions to guard against poisoning by the drug be taken.

INFECTIOUS MASTITIS OF GOATS IN GEORGIA, U.S.S.R. A. I. Toidze. *Veterinaria,* No. 7, 1954.

GOAT keeping forms a considerable part of the rural economy of Western Georgia and large herds are maintained on the rough mountain pastures where they subsist and thrive on a diet largely derived from thorn bushes and plants, usually classified as weeds. Among the best milkers of the Megrelski breed the annual milk yield often reaches about 800 to 900 litres.

Infectious mastitis causes considerable loss to the goat farmers, and in certain areas the disease precludes the keeping of goats. When the infection attacks a flock somewhere in the region of 95 per cent of the animals are involved and the losses amount to between 70 and 100 per cent; those that recover show an induration and atrophy of the udder and are no longer a commercial proposition; among the infected animals the death rate reaches 60 per cent. It would appear from a number of experiments that the disease is carried by animals who have recovered from a previous attack although they themselves are not susceptible to taking the disease a second time. It is essentially a disease of the lactation period, and dry nannies and those with lambs are not infected. Under natural conditions three to four days is the incubation period, and the disease runs its course from four to six days.

The infectious mastitis of goats takes the form of an acute purulent mastitis with abscess formation. Usually only one half of the udder is infected, it becomes enlarged and very painful, and the skin over it is inflamed; there is a rise in temperature and loss of appetite. Gangrene may supervene, patches of necrosis may appear on the base of the teats, and the animal may bite off the gangrenous nipples and a purulent and slow-healing wound is left behind. At post-mortem a purulent peritonitis, gangrene of the udder and the adjacent muscles is revealed. There are signs of a general sepsis—inflammation of the kidneys, liver and heart; there is also extravasation of blood into the mucous membranes.

Pure cultures of the causal organism may be obtained from the udder, from the blood during the rise in temperature, from the kidneys, liver, etc. The organism is a gram negative rod. 0.7 to 2.4 long, aerobic, has no capsule and does not form spores; it grows well on the usual media at 37 deg. Centigrade and at *pH* of 7.2. It would appear to be similar in many ways to *Pseudomonas pyocyanea.* This condition may be differentiated from the infectious mastitis of sheep and goats by its attacking lactating animals only and by the absence of arthritis and keratitis. As regards curative measures results have been obtained by a formolised vaccine and gramicidin C has been beneficial when its use has been resorted to early in the disease.

The large and low-hanging udder of this Megrelski breed of goats is thought to have some influence on the susceptibility of these animals to this condition.

R.H.A.M.

REVIEWS

VETERINARY NECROPSY PROCEDURES, edited by T. C. Jones and C. A. Gleiser, 1954. London: Sir Isaac Pitman & Sons, Ltd. Price 50s. net.

IN planning a textbook on post-mortem technique, it must be difficult to decide where to place the limits between what should be included as essential and what should be excluded as superfluous. When making a post-mortem examination, the veterinary surgeon, in trying to arrive at a precise diagnosis by the shortest and yet the surest route, must be able to bring to bear a very considerable knowledge of normal and morbid anatomy. It would be possible to make out a case for a really compendious handbook, which would, for example, include lists of weights and sizes of different organs, would note the common normal variations of anatomical structure, and would mention all possible morbid anatomical findings for each part of the body. On the other hand, it might be argued that the only way to learn post-mortem technique is under instruction from a competent teacher, and that textbooks of anatomy and pathology already cover the ground apart from the actual technical methods.

This book, on the whole, seems to strike a fair balance, tending, perhaps, to be too brief rather than too long in certain parts. Its great value lies in a series of lucid and well-illustrated descriptions of actual post-mortem examination procedures. The book is sponsored by the Armed Forces Institute of Pathology and the American Veterinary Medical Association, and edited by two Registrars of the Registry of Veterinary Pathology, T. C. Jones and C. A. Gleiser. The text matter describes the technique of opening the various body cavities, and removing the different organs, and their subsequent examination.

The first chapter in the book is largely a reprint of an editorial from an American veterinary journal on the value of the post-mortem examination. The second sentence—which states that the last substantial publication on the subject appeared in 1918—will hardly be acceptable to any pathologist who can read German. The next chapter reviews general principles of post-mortem technique, and then follow chapters on the autopsy technique for the horse (Hilton Smith), ox and sheep (P. Olafson), pig (L. M. Roderick), dog and cat (D. L. Coffin), fowls and other birds (E. L. Stubbs), laboratory animals (C. Olson), and wild animals (L. J. Goss). Of these chapters, that by Coffin is particularly good. A key chapter in the book is that by T. C. Jones, in which a suggested form of post-mortem report is discussed, and useful lists of terms descriptive of shape, colour and so on are given. Many students (and graduates) can well detect morbid anatomical changes, but are sometimes at a loss for the right words to describe them, and this chapter should be very helpful. Then R. Randall discusses the selection and preparation of specimens for laboratory examination from the point of view of the type of examination required. H. J. Griffiths gives a very useful account of the collection and preservation of parasitological specimens, and H. R. Seibold suggests what particular specimens should be submitted for examination when certain diseases are suspected.

The book is addressed to an American audience, but it can be recommended to students and graduates over here. A particularly valuable feature of the book are the many drawings illustrating the various stages in different autopsies and, with their aid, it should be possible for a post-mortem examination to be tackled with confidence. It is to be regretted that the high price of the book may tend to restrict what would otherwise perhaps be a very wide sale.

FERTILITY AND INFERTILITY IN THE DOMESTIC ANIMALS, by J. A. Laing, Ph.D., B.Sc., M.R.C.V.S., 1955. London: Balliére, Tindall & Cox. Pp. XIII and 256, 27 illustrations and 27 plates. Price 35s.

THIS book contains an up-to-date survey of its subject, and can be thoroughly recommended to veterinary students, veterinary surgeons and livestock breeders.

Section I deals with the anatomy and physiology of the various reproductive systems, the diagnosis of pregnancy and the techniques of artificial insemination.

Section II describes the various disorders and diseases that may affect fertility, and discusses their ætiology, diagnosis and treatment.

The title of the book is misleading for, although the author states that his book covers the cow, horse, sheep, pig and dog, information on the dog is scanty, and the cat is not even mentioned. If the word "Farm" were substituted for "Domestic" it might be a more fitting description.

The illustrations and plates are excellent, and the print and binding a credit to the publishers.

The comprehensive lists of references will be of great assistance to students and indicate the great amount of work that has been reviewed.

The author can be complimented on producing what may become a standard textbook on the subject.

BRITISH POISONOUS PLANTS, by A. A. Forsyth, M.R.C.V.S., D.V.S.M. Bulletin No. 161, Ministry of Agriculture and Fisheries. London: H.M. Stationery Office. Pp. 116, 9 plates. Price, 6s. 6d.

THIS bulletin, which replaces Bulletin No. 75, "Poisonous Plants on the Farm," fills a gap that has existed for too long in the veterinary literature. After dealing in general terms with poisonous plants, their toxic principles, and the effects they produce, the author devotes the major part of the book to a detailed survey of the more important poisonous plants arranged according to their botanical classification. In addition, there are sections dealing with moulds and ergot, the larger fungi, plants of suspected toxicity, and plants that taint milk. There is also a useful glossary of technical terms. After giving the main characteristics by which a plant may be recognised, the author deals with its effects on the body, the symptoms produced, and the post-mortem findings. Advice as to first-aid treatment is given in most cases.

The book considers poisoning in domestic animals rather than in man. This is understandable as, happily, the days are now past when whole families

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were driven by poverty into making unwholesome meals of *Calthra palustris* and *Œnanthe crocata*.

Although the author states in his preface that the book is written primarily for the stockowner and countryman, there is no doubt that it will prove of great use to the veterinary reader. Furthermore, the large number of references quoted should prove very valuable to the research worker in this field.

It is perhaps invidious to criticise such a welcome publication, and in suggesting that one would have liked more illustrations and a better binding, one realises that these could only have been supplied at a greatly increased cost.

ESSENTIALS OF HORSEMANSHIP, by Brigadier-General J. F. Lamont. London. Constable and Company, Limited. Price 12s. 6d. net.

THIS book is "out of the run" of the usual texts on horsemanship and riding.

Brigadier Lamont is a man of few words but he has a unique appreciation of the essentials of the theory and practice of equitation and he is able to describe these in words which can be understood by a novice and be respected and appreciated by an accomplished horseman.

Any horseman will be a better horseman after he has digested this book. The illustrations have been selected with great care, they are not so numerous as to be confusing, and the reader can turn to them time after time and with profit.

ITEMS OF VETERINARY INTEREST

DR. SADEK M. JEWAD, Director General of Iraqi veterinary services, is at present on a visit to Britain as a guest of the British Council in order that he may visit our veterinary colleges, research centres and agricultural shows.

IT has now been confirmed that a British brown hare (sp. *Lepus Europæus occidentalis*), killed by a dog at Wilton, Wiltshire, recently, was suffering from myxomatosis. This confirms the findings in France that in rare instances the hare may be affected but it is emphasised that there is no evidence that the disease affects other animals or human beings.

On February 11 the Ministry of Agriculture and Fisheries confirmed that that the carcase of a hare found in Northern Ireland was affected with myxomatosis. The hare was a mountain hare (sp. *Lepus timidus*) also known as the blue or varying hare. This was the only other occasion on which myxomatosis had been confirmed in a hare in the United Kingdom. *M.A.F.F. 71/55.*

THE condition among cattle and sheep, known as hypomagnesæmia, is discussed in a pamphlet issued by the Advisory Department, The British Oil and Cake Mills Ltd. The object is to clarify the symptoms of the disease which is stated to be on the increase in Britain. Suggested means of treatment and prevention are by use of the company's mineral supplements of magnesium oxide and calcinated magnesite. A copy of the brochure which is informative and a useful reference for diagnosis will be supplied on request.

THE Irish Government have been obliged to restrict by nearly one-third the number of acres allotted this year to beet production owing to the appearance of eel worm in some parts of the country during 1954. Most of this year's crop has now been sown and its condition is satisfactory.

WATSON & SONS (ELECTRO-MEDICAL) LTD. have recently designed and built a high-power X-ray unit for use at the Animal Health Trust's Equine Research Station at Newmarket. It comprises a high tension transformer with output of 120 kV, 400 mA, coupled to a high-power rotating anode X-ray tube. The object is to obtain the maximum penetration in the shortest possible time to facilitate radiography of large and nervous animals and dispense with anæsthetics where possible. It is hoped that with powerful apparatus of this kind it may be possible to extend radiological investigation of large animals to diagnosis of disorders of the digestive system and lungs.

To simplify injection work and reduce pain incurred by animals a new hypodermic needle has been introduced by Chas. F. Thackeray Ltd. This has a diamond-shaped point which passes more easily through the animals' tissues and the bevelled shaft reduces friction and prolongs the life of the needle. The "Diapoint," as it is called, is another evidence of the way manufacturers are constantly devoting their resources to improve the veterinary surgeon's equipment.

NOTICE

THE introduction of a 100 per cent mineral pellet as from June 1 is announced by British Glues and Chemicals Ltd. The formula of these mineral pellets is identical to that of the manufacturer's powdered minerals and no additive or diluting agent is used.

The pellets are small, hard cylinders about $\frac{3}{8}$ in. long. It is claimed that they are easy and pleasant to use, particularly as they are free from dust. Mixing minerals with dairy cakes, cubes or nuts is said to be simplified by the use of these pellets.

OBITUARY

WE regret to learn of the death on May 18, at the age of 39 years, of Mr. A. H. Milne, M.R.C.V.S, Chief Veterinary Research Officer, Mpwapwa, Tanganyika. His death occurred as a result of a plane crash while he was flying to Abyssinia on an assignment for F.A.O. before returning to Britain on leave. He was to assist in the organisation of veterinary education in Abyssinia and had been loaned by the Colonial Office for this purpose. Education was of great personal interest to Mr. Milne and he was instructor and examiner in Anatomy at the University College of East Africa, Makerere. He was very popular though of an unassuming and quiet nature; his death is a severe loss to the Colonial Veterinary Service and we tender our sincere sympathy to his wife.

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